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VOLUME 47 MAY, 1934 NUMBER 5

SPREADING IN A MONOMOLECULAR FILM

A METHOD FOR STUDYING BIOLOGIC PROBLEMS

PROF. DR. E. GORTER

LEYDEN, THE NETHERLANDS

I have been occupied with the following ideas and experiments for several years. Therefore the subject is of such great interest to me that I may not see with a clear eye all the deficiencies of the method described here, and I may overestimate its value.

Langmuir, one of the most famous physicists, found that some substances can be spread on a watery surface in a layer that is only one molecule thick. Palmitic acid, for example, may be dissolved in petroleum ether, and a small, accurately measured amount of the solution placed on the surface of water in a large tray. After evaporation of the solvent a barrier can be moved in such a way that the surface is gradually diminished in size and the molecules of the fatty acid are brought together. When the molecules have all come into contact with each other the surface tension suddenly diminishes. This can easily be shown by determining the surface tension, e. g., by the method of du Nouy (figs. 1 and 2) or, better, by using the apparatus of Langmuir (figs. 3 and 4).

The reason why the molecules of some substances spread is that one part of each molecule is soluble in water and another part insoluble. Fatty acids spread because the carboxyl group is soluble and the paraffin chain, if it is of sufficient length, insoluble.

For the quantitative study of the size of a molecule under the conditions set forth, the curve shown in figure 5 is used, in which the surface per molecule (obtained by dividing the spread per mol of the substance by Avogadro's number, 6.06×10^{23}) is plotted against the pressure on the strip of the balance in dynes per centimeter. Extrapolation to a pressure of zero gives the size of the molecule in a transverse section.

If the foregoing explanation of the partial solubility of the molecules is correct, all the fatty acids should occupy the same surface, whatever the length of the chain may be, and this is what is actually found when they are studied under the same conditions.

Read at a meeting of the American Pediatric Society, Washington, D. C., May 9, 1933.

The biologist can apply this method to a large series of problems, for living matter is largely built up of spreading substance. Not only fatty acids, fats, lipoids and sterols (fig. 6), but even most proteins belong to this group of substances. The last-mentioned fact is striking on first consideration, because a protein substance dissolved in water should not be able to spread.

The explanation of this apparent contradiction lies in the fact that a protein molecule has a different structure when in solution than when spread on a watery surface.

Before considering further the problem of the spreading of proteins I shall describe some possible applications of the spreading of lipoids.

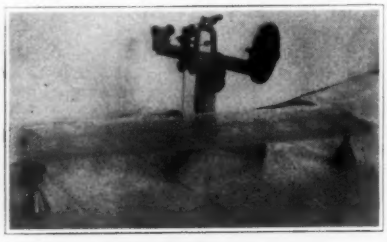


Figure 1

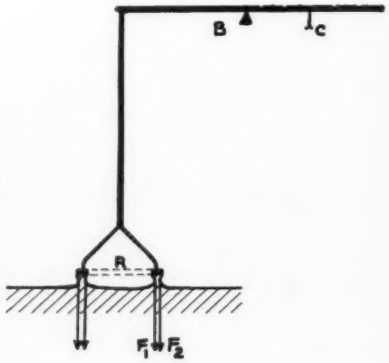


Fig. 2

Fig. 1.—Du Nouy's tensiometer.

Fig. 2.—Diagrammatic representation of du Nouy's apparatus. The platinum ring, *R*, hanging on one side of some kind of balance, schematically represented by the knife of the balance *B*, can be lifted from the surface. The surface forces F_1, F_2, acting on the balance, can be counterbalanced and measured by weights *C*.

The following example gives an idea of the importance of the fact that a substance can spread only when there exists in its molecule a certain balance between polar and nonpolar groups.

In the first members of the series of fatty acids with an increasing number of CH_2 groups the soluble part has an easy task in pulling the insoluble part—small as it is—into the water. But gradually, with the increasing length of the chain, the force acting in the reverse direction becomes greater and greater. When the number of carbon atoms is ten, the pulling force of the carboxyl group is not strong enough to overpower the insoluble group, and spreading results as a compromise. With eight carbon atoms in a fatty acid there is no spreading. In the series of fats the same events can be traced, but, owing to the linking of the three fatty acids in one molecule, spreading is observed when

the number of carbon atoms is six and is complete in the next member of the series with eight carbon atoms (fig. 7).

This permits an interesting application of the present method to a biologic problem, namely, the study of a lipolytic enzyme.

If one adds to an emulsion of tricaprylin a solution containing a lipolytic enzyme the fat will be partially split into caprylic acid, the fatty acid with eight carbon atoms.

By an extraction of the tricaprylin and lipase solution, before and after completion of the splitting process, with a solvent for fat and fatty acids one obtains a quantitative estimation of the number of molecules of fat that have been split into fatty acid by spreading both

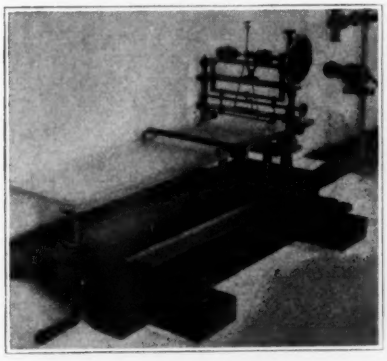
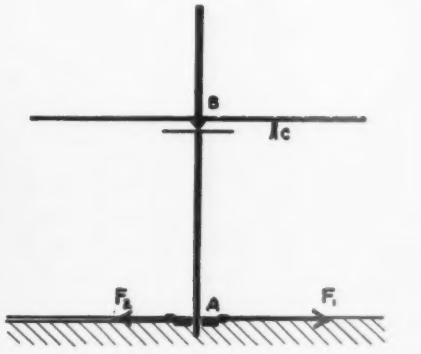


Figure 3



LANGMUIR-BALANCE

Figure 4

Fig. 3.—Langmuir's balance.

Fig. 4.—Diagrammatic representation of Langmuir's apparatus. A floater, *A*, forming part of a balance with the knife at *B*, will move under the influence of the difference in surface tensions, F_1, and F_2 (the diminution of F_1 to F_2 is caused by some spreading substance). This movement is prevented by placing weights *C* on the balance. In this way the difference $F_1—F_2$ can be measured.

of the extracts in a monomolecular layer. In table 1 I present the results of such an experiment.

One advantage of this method is that very small amounts of different substances can be determined, because one employs essentially a microdosage. This advantage can be appreciated by pediatricians, who are often handicapped by the difficulty of getting sufficient material for estimations.

It is obvious that for a quantitative estimation of fats and lipoids in a Bloor extract of blood or serum, the spreading method can be used with great success. The small amount of blood necessary for these estimations is shown by the fact that, as a rule, 0.01 mg. can cover a sur-

face of from 50 to 100 sq. cm., an area in which determinations yield very slight experimental error.

I shall not enter into further detail concerning this method of estimation, which, though perhaps of some advantage, is not essentially

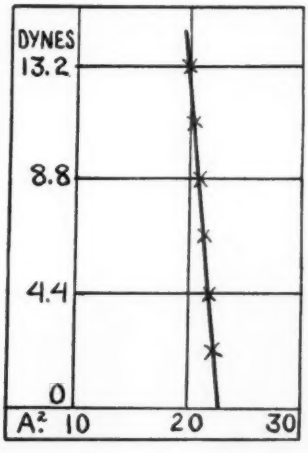


Fig. 5.—Curve of surface area of palmitic acid from a soap solution spread on hundredth-molar hydrochloric acid, p_H 2. A.² indicates square angstroms. (This determination was made by Dr. Grendel.)

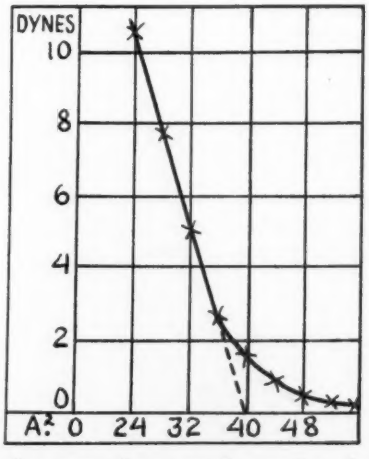


Fig. 6.—Curve of surface area of ergosterol, spread on thousandth-normal hydrochloric acid. A.² indicates square angstroms.

different from existing procedures and is not easily employed in every laboratory.

I perfer to describe a different application of spreading in a monomolecular film. It seems reasonable to expect that, in living matter,

spreading substances are present in a layer that is one molecule thick. As a relatively simple form of living matter, the red blood cells of mammals were chosen for investigation, as these have lost the most complicated part of a cell, the nucleus.

The answer to the following question was sought: Is the surface of the red blood cell covered by a layer of lipoids one molecule thick, or

TABLE 1.—*Determination of Lipase by Spreading Method*

	Immediate Extraction	Extraction After 1 Hour at 40 C.
Tricaprylin	10.2	10.4
Serum	4.6	4.0
Serum and tricaprylin	14.8	12.0

TABLE 2.—*Correlation Between the Surface of the Red Blood Cells and the Surface of the Total Lipoids in Man and the Goat* *

Source	Amount, Cc.	Surface of 1 Cell in 10^{-8} Sq. Cm.	Total Surface of Red Cells in 10^4 Sq. Cm.	Surface of Lipoids in 10^4 Sq. Cm.
Goat	1	20.1	0.33	0.66
	1	20.1	0.33	0.69
	10	17.8	3.34	6.1
	10	17.8	3.34	6.8
	1	17.8	0.33	0.63
Man	1	99.4	0.47	0.92
	1	99.4	0.47	0.89

* Determinations were made with Dr. Grendel.

TABLE 3.—*Correlation Between the Surface of the Red Blood Cells and the Surface of the Total Lipoids in the Sheep, the Dog and the Guinea-Pig* *

	Surface of 1 Cell in 10^{-8} Sq. Cm.	Total Surface of Red Cells in 10^4 Sq. Cm.	Surface of Total Lipoids in 10^4 Sq. Cm.
Sheep	29.8	0.30	0.62
	98	0.78	1.55
Dog	90	0.62	1.22
	101	0.98	1.96
	124.8	0.54	1.08
Guinea-pig	89.8	0.52	1.02
	100.8	0.57	1.16

* Determinations were made with Dr. Grendel.

(for this is the only fact that can be determined directly) is there any simple relation between the measured surface of red blood cells and the lipoids that have been extracted from them and spread on the surface of water in a Langmuir tray?

The first difficulty to surmount was: How can one measure the surface (in a layer one molecule thick) of the lipoids of red blood cells from, say, 1 cc. of blood? The following procedure was used: The red

blood cells of 1 cc. (or 10 cc.) of blood are washed with saline solution, and then extracted with different solvents (Bloor's alcohol and ether mixture, acetone followed by alcohol, ether and petroleum ether). By spreading the extracts on a watery surface, the total surface of these lipoids can easily be calculated.

TABLE 4.—*Correlation Between the Surface of the Red Blood Cells and the Surface of the Total Lipoids in Man and the Rabbit* *

	Surface of 1 Cell in 10^{-8} Sq. Cm.	Total Surface of Red Cells in 10^4 Sq. Cm.	Surface of Total Lipoids in 10^4 Sq. Cm.
Man	123	0.64	1.2
	90	0.18	0.35
Rabbit	92.5	0.54	1.07
	74.4	0.49	0.96
	180	0.56	1.18
	92.4	0.51	0.92
	92.5	0.55	1.1

* Determinations were made with Dr. Grendel.

TABLE 5.—*Surface Structure of Red Blood Cells of Sheep* *

	Percentage of Surface
Cholesterol	36
Cephalin	50
Sphingomyelin	13

* Determinations were made by Dr. Grendel.

TABLE 6.—*Spread of Fats and Lipoids* *

	Spread per Molecule in Square Angstroms	Spread per Mg. in Sq. M.
Lecithin	115	0.86
Cephalin	116	0.87
Sphingomyelin	46	0.35
Cholesterol	38.5	0.6
Palmitin	51	0.38
Olein	126	0.9

* Determinations were made with Dr. Grendel.

The next point is: How can the surface of all the blood cells in 1 cc. of blood be determined? To obtain this figure the formula $2\,D^2\,N$ (Knoll) was used. (D is obtained by drawing several hundred red blood cells from a blood smear with the aid of a Zeiss drawing prism; N is determined in the usual way, in a counting chamber.) Tables 2, 3 and 4 give some of the results obtained in this way.

There exists a simple relationship between the two values: the surface of the red blood cells and the surface of the total lipoids form the same amount of blood. It is almost without exception 1:2→. As stated by Professor Ehrenfest, whose encouragement in this work

has been of great value to me, the most probable explanation is the following: The lipoids are estimated at an interphase between water and air, whereas a red blood cell has a wall that is limited on both sides by a watery medium; therefore two layers are to be expected, and not one.

My collaborator, Dr. Grendel, has studied the different lipoids in the red blood cells in more detail. He found the composition shown in table 5, expressed in surface units.

TABLE 7.—*Influence of Calcium on the Spread of Lipoids* *

	No Calcium: Area, Sq. M.	Calcium, 5 Mg. per 100 Cc.: Area, Sq. M.	Calcium, 11 Mg. per 100 Cc.: Area, Sq. M.
Sheep	0.68	0.69	0.62
	0.74	0.79	0.73
	0.87	0.91	0.79
	0.92	0.91	0.83
Goat	0.74	0.80	0.69
	0.76	0.76	0.67

* Determinations were made with Dr. Grendel.

TABLE 8.—*Spread of Serum Proteins (Man, Rabbit, Goat, Cow)* *

	Albumin			Globulin		
	Sq. M. per Cc.	Per Cent	Per Mg.	Sq. M. per Cc.	Per Cent	Per Mg.
Man	47	5.2	0.90	21	3.4	0.62
	42	4.6	0.91	24	4.0	0.60
	41	4.5	0.91	29	4.7	0.62
	29	3.2	0.91	40	6.6	0.61
Rabbit	45	4.9	0.92	15.5	2.7	0.59
	40	4.3	0.93	21	3.5	0.60
	36	3.9	0.92	12.5	2.1	0.60
Goat	41	4.5	0.91	16	2.7	0.60
	40	4.3	0.93	22	3.6	0.61
	36	4.0	0.90	16	2.6	0.61
	38	4.2	0.90	16.5	2.7	0.61
Cow	31	3.4	0.91	36	6.0	0.60
	28	3.1	0.90	19	3.1	0.61

* Determinations were made with Dr. Grendel.

It is obvious that in every method that allows an estimation of these substances in milligrams the amounts can easily be recalculated in square centimeters by making use of table 6, in which the spreading of fats and lipoids is given for 1 mg. of each substance.

This study of red blood cells is an example of a possible application of this new method to the elucidation of the structure of living cells. It is interesting to speculate further and to suppose that this lipoid also exists in other cells, but I prefer not to go too far into a hypothetic field.

There is only one point that is important to mention in this respect: the influence of calcium ions on the size of the molecules of lipoids

from red blood cells. If calcium is added to the water in the tray, in the amount of 11 mg. per hundred cubic centimeters of fluid, the area occupied by the molecules is diminished (table 7).

It is possible to believe in the general importance of this influence of bivalent ions, because we have observed it in proteins, and it is perhaps not too hypothetic to bring these facts into relation with the influence of calcium ions on permeability.

TABLE 9.—*Spread of Serum Proteins (Sheep, Horse)* *

	Albumin			Globulin		
	Sq. M. per Cc.	Per Cent†	Per Mg.	Sq. M. per Cc.	Per Cent‡	Per Mg.
Sheep	36	4.0	0.90	17	2.8	0.61
	40	4.4	0.91	25	4.0	0.62
	47	5.1	0.92	19	3.3	0.58
	33	3.6	0.92	28	4.6	0.61
Horse	22	2.3	0.96	39	6.4	0.61
	37	4.1	0.90	23.5	3.8	0.62
	29.5	3.2	0.92	36	6.0	0.60
	29	3.2	0.91	46	7.5	0.61
	31	3.4	0.91	34	5.6	0.61

* Determinations were made with Dr. Grendel.
† Gravimetrically.
‡ By subtraction: total protein minus albumin on tenth-molar hydrochloric acid.

TABLE 10.—*Determinations of Serum Proteins in Large Series* *

Patient; Condition; Age	Albumin, per Cent	Globulin, per Cent	Total, per Cent
Breast-fed; 3 months	5.0	2.8	7.8
Breast-fed; 6 weeks	4.4	2.5	6.9
Breast-fed; 6 weeks	4.6	2.5	7.1
Athrepsia; 9 weeks	3.7	1.9	5.6
v. d. H. Sept. 13	4.8	3.4	8.2
Sept. 14: dehydration	4.7	2.4	7.1
Sept. 20: 6 months	4.0	2.8	6.8
Nephrosis; 10 years	1.6	4.1	5.7
	1.7	3.5	5.2
Active tuberculosis; 1 year	2.9	5.4	8.3
	4.3	5.7	10.0

* Determinations made with J. Munk.

There is another aspect of the monomolecular film and its importance in biology. Among the different substances that can produce hemolysis there are several belonging to the class of spreading substances. One group is especially interesting because its members have a varying tendency to spread, owing to the greater or lesser number of dextrose molecules; these substances are the saponins.

Through the courtesy of the late Dr. van der Haar, of Utrecht, I investigated the spreading of different pure saponins. The best spreading was obtained with parillin. The size of the surface occupied by 1 mg. of this saponin in a monomolecular film was therefore easily determined (fig. 8). This made possible the following experiment:

In a known amount of red blood cells we measured the total surface, as I have described. Then the minimum amount of parillin that produced total hemolysis was determined; this amount was also expressed in surface units. When the surface of the red blood cells was placed in

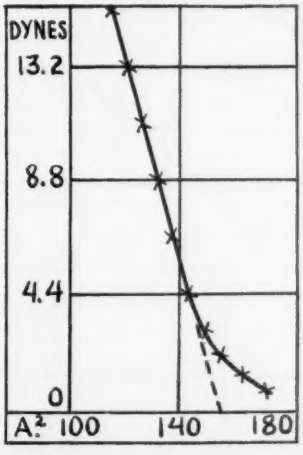


Fig. 7.—Curve of surface area of tricaprylin, spread on tenth-molar hydrochloric acid, A 1. A.² indicates square angstroms. (This determination was made by Dr. Grendel.)

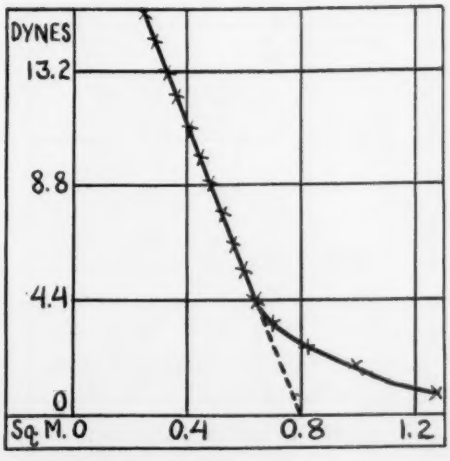


Fig. 8.—Curve for spreading of parillin on nine-tenths normal sodium chloride at 20 C. (This determination was made by Dr. Grendel.)

a monomolecular film against the surface of the parillin, that is, in just sufficient amount to produce complete hemolysis, we obtained the relation 1 : 1, as is clearly demonstrated by figure 9.

As I have already said, many proteins can be induced to spread on a watery surface. Some of them spread well; others are too soluble. Gelatin, the protamines and fibrinogen spread poorly, whereas most other proteins spread excellently.

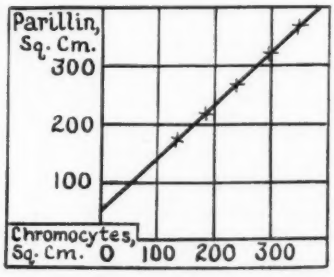


Fig. 9.—Hemolysis by a saponin (parillin).

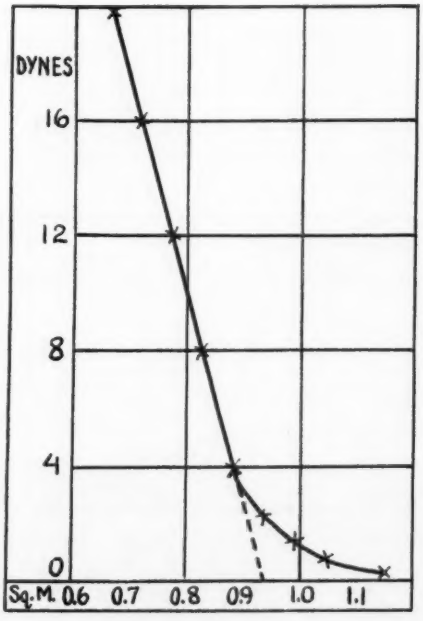


Fig. 10.—Curve for spreading of ovalbumin on tenth-molar hydrochloric acid, p_H 1.

Figure 10 shows the surface tension curve of ovalbumin on tenth-molar hydrochloric acid, and figure 11 a similar spreading of zein at p_H 5.

The spreading of proteins is often obtainable only by taking account of hydrogen ion concentration (fig. 12), the influence of which is

demonstrable only if a solution containing little electrolyte is used. The spreading is maximal only at the iso-electric point and in a strongly acid or alkaline solution.

One important point is that a protein film has a very small thickness, from 6 to 7.5 angstroms, although the molecule is as large as 44,000 cubic angstroms. This leads to the view that on a watery surface the molecule is spread out as a flat platelet.

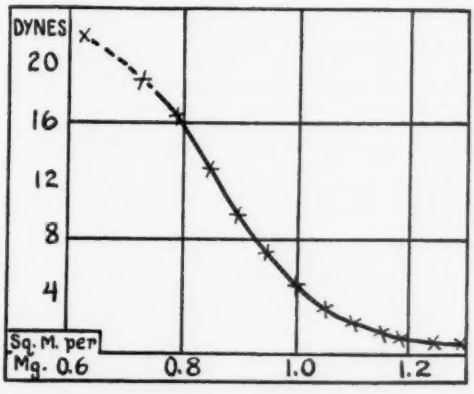


Fig. 11.—Curve for spreading of zein on acetate buffer (three-hundredth molar solution, p_H 5).

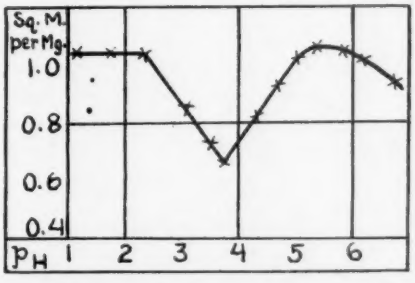


Fig. 12.—Influence of p_H on the spreading of zein.

It is clear that when conditions are chosen accordingly, this method may enable one to determine quantitatively the amount of protein in a solution. To this end it is necessary only to find the number of square centimeters that corresponds to, say, 1 mg. of each protein. This factor has been determined in my laboratory for the proteins in blood serum, as is indicated in tables 8 and 9. By making use of this factor it is possible to calculate inversely from a certain spreading surface how many milligrams are contained in a serum.

Table 10 gives some results of a very large series of determinations showing that the typical variations in the protein content of the blood

serum and its distribution over the two fractions can be found by using this method.

In athrepsia the total protein content, especially the globulin fraction, is low, whereas the low protein content of serum in a case of nephrosis is due to a decrease in the albumin fraction.

In dehydration a high total protein content is noted, and in active tuberculosis a high globulin fraction.

With proteins the enzyme action can be controlled by means of spreading because the split products resulting from the tryptic digestion of spreading proteins do not spread. This has been carefully studied by Dr. Grendel, who has shown that a reduction of the area occupied

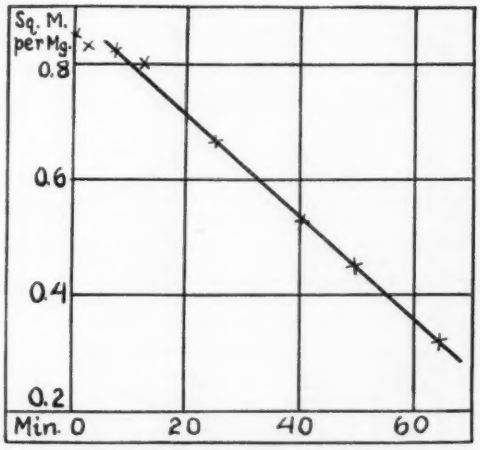


Fig. 13.—Influence of ultraviolet light on the spreading of insulin on thousandth-molar acetate solution, p_H 4.5.

by one molecule is a very sensitive indicator of the digestive action of the trypsin.

The same observation holds good with regard to the control of the deleterious effect of ultraviolet rays on proteins. This can easily be demonstrated in the following experiment: Insulin dissolved in acid (p_H 2.4) is spread on a watery surface and then irradiated with a powerful vapor arc lamp in quartz, placed above the surface of the tray. The surface diminishes gradually in the course of one hour (fig. 13), through the gradual disappearance of the spreading protein from the surface.

By this brief enumeration of facts and experiments I have tried to present a general view of the possibilities that the method described offers to the biologist. I am firmly convinced that the points mentioned constitute only a small part of the knowledge to be gained from the use of this spreading method.

DISCUSSION

Dr. L. Emmett Holt, Jr., Baltimore: I might say that three years ago I was at Dr. Gorter's clinic in Leyden and saw his apparatus. We have since built one in Baltimore and have used it for the micro-estimation of fat. I believe that it is the most satisfactory micromethod for the estimation of fat that is available.

Dr. A. A. Weech, New York: I should like to be among those to thank Dr. Gorter for a most fascinating presentation, and to tell him that his love for his subject has been sufficient to inoculate a number of us who have listened to his address.

The possibilities of the method, although I have had no experience with it, come to me with peculiar force because we have been struggling with the problem of trying to measure the amount of protein in biologic fluids that contain very small quantities, particularly edema fluids and lymph. The ordinary methods now available generally imply the determination of nonprotein and total nitrogen, the difference being calculated as protein. All sorts of attempts, most of them unsatisfactory, have been made to get away from the necessity of calculating by difference.

Dr. Gorter's technic presents a new way of accomplishing this purpose, which I think should be of tremendous help.

Dr. E. Gorter, Leyden, The Netherlands: May I answer Dr. Weech? I fear that it would be difficult to get results with very dilute solutions, but there is a paper in the *Chinese Medical Journal* indicating that the author has used such solution. We have used only the concentrated solutions. With them results are obtained in a short time. If one uses the diluted solution, one will have to wait hours before reliable results can be obtained.

INCIDENTAL HYPERGUANIDINEMIA AS A CAUSE OF CLINICAL TETANY

KATHARINE DODD, M.D.
AND
A. S. MINOT, PH.D.
NASHVILLE, TENN.

The majority of evidence indicates that the clinical condition known as tetany results from a lack of the physiologic effect of calcium ions. The types of tetany in which the relationship to a deficiency of calcium is most apparent are parathyroid tetany and so-called "infantile tetany." In these conditions the most constant chemical change in the blood is a marked reduction in the calcium content. Tetany may also accompany a condition of alkalosis in which the total calcium is not reduced, but in which there is a lack of effective or ionized calcium. The same symptoms can be produced experimentally by the injection into the blood stream of solutions of sodium phosphate, which cause a reduction in the calcium of the blood and also by the injection of citrates or oxalates which form unionized complexes with the calcium of the blood. It has been argued by some investigators [1] that the essential feature in the production of tetany is an increase in the sodium: calcium ionic ratio in the blood. Such a change obviously occurs when the calcium is markedly reduced and the concentration of the antagonistic sodium ion is normal. The injection of so inactive a salt as sodium chloride in amounts sufficient to increase greatly the concentration of sodium ions in the blood may produce tetany even in the absence of a markedly reduced concentration of calcium.[1a] It seems possible that a similar relative lack of effect of the calcium ion might be produced by the presence in the body fluids of an excess of any substance physiologically antagonistic to calcium. It is not our purpose in this paper to discuss the more common types of tetany associated with hypocalcemia or alkalosis. We wish instead to present several instances in which tetany apparently resulted from the presence of an excess of a substance which exerts a physiologic action antagonistic to that of calcium ions.

From the Department of Pediatrics, Vanderbilt University Hospital.

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1. (*a*) Greenwald, I.: Observations on the Effect of Intravenous Injections of Some Sodium Salts with Special Reference to the Supposed Toxicity of Sodium Phosphate, J. Pharmacol. & Exper. Therap. **11**:281, 1918. (*b*) Tisdall, F. F.: The Influence of the Sodium Ion in the Production of Tetany, J. Biol. Chem. **54**:35, 1922.

Name, Age	Diagnosis	Date	Symptoms of Tetany	Electric Excitability, Ma.	Roentgenogram of Extremities	Mg. per 100 Cc.			Serum Protein, per Cent	Carbon Dioxide		Comment
						Guanidine	Calcium	Inorganic Phosphate		p_H	Content, Vol. per Cent	
P. W., 1 year	Influenza; bronchopneumonia	11/ 9/32	Chvostek sign; rigidity; carpopedal spasm	CCC. 1.5; ACC. 4.0; AOC. 4.0; COC. 4.2*	No rickets	0.66	10.4	3.4	6.4	....	....	No clinical evidence of or reason for alkalosis; tetany relieved by the administration of calcium gluconate intravenously
		11/14/32	None	CCC. 3.5; ACC. 7.0; AOC. 6.5; COC. 5.2	.....	0.38	12.5	5.6	7.4	....	....	Recovered
H. L., 8 months	Influenza	11/ 3/31	Stiff face; crow; carpopedal spasm	.....	.....	0.61	9.0	6.7	...	7.30	41.4	Tetany promptly relieved by the administration of calcium gluconate intravenously
		11/10/31	None	.....	.....	0.32	11.6	6.0	...	....	....	Recovered
B. D., 14 months	Bronchopneumonia	12/21/32	Crow; restlessness; irritability	CCC. 3.2; ACC. 3.0; AOC. 4.9; COC. 3.8	No rickets	0.75	8.8	3.5	5.2	7.39	58.6	Tetany relieved by the administration of calcium gluconate intravenously
		12/22/32	None	CCC. 3.5; ACC. 5.5; AOC. 8.0; COC. 6.5		....	....	...	...	....	....	
		12/23/32	None	CCC. 3.5; ACC. 6.2; AOC. 7.4; COC. 6.7	.....	....	....	...	...	....	....	Taken home against advice; died of pneumonia
B. B., 1 year	Paratyphoid fever (B. paratyphoid B); pyelitis; diarrhea	6/ 7/32	Convulsions; stiff face; carpopedal spasm	.....	.....	0.62	9.0	3.3	5.1	7.37	43.3	Tetany relieved by the administration of calcium gluconate intravenously; died of infection
D. A., 2 years	Lobar pneumonia	3/ 9/32	Carpopedal spasm; irritability; stiff face	.....	.....	0.64	10.1	2.3	7.4	7.52	54.6	Tetany disappeared gradually with intramuscular medication with calcium gluconate
		3/19/32	None	.....	.....	0.37	....	...	...	....	....	Recovered
R. M. B., 18 months	Influenza; bronchopneumonia	12/21/32	Chvostek sign; Trousseau's phenomenon	CCC. tetanus ACC, 3.5; AOC. 3.8	No rickets	0.64	9.8	4.0	6.6	7.38	60.0	Tetany relieved temporarily by each intravenous dose of calcium gluconate
		12/22/32	Chvostek sign; carpopedal spasm	CCC. 1.8; ACC. 3.9; AOC. 3.7; COC. 1.8	.....	....	....	...	...	....	....	Died 12/24/32
E. G., 17 months	Chronic meningococcic meningitis	3/21/31	Stiff face; carpopedal spasm	CCC. 2.0; ACC. 1.7; AOC. 2.0; COC. 1.5	No rickets	0.62	10.6	5.5	...	....	57.0	Tetany relieved by medication with calcium gluconate
		4/ 2/31	None	.....	.....	0.47	11.1	...	...	....	....	Recovered after treatment with antimeningococcic serum
M. N. 14 years	Pellagra; ulcerative colitis	8/ 8/32	Stiff face; unable to open jaws	.....	.....	1.13	....	...	...	7.28	44.0	Tetany relieved by the intravenous injection of calcium gluconate
		8/10/32	Same	.....	.....	0.94	11.0	...	...	....	....	
		8/18/32	None	.....	.....	0.39	....	...	...	....	....	Slow recovery from pellagra
M. W., 59 years†	Bronchopneumonia; pellagra (?)	3/23/31	Carpopedal spasm; rigidity; convulsions	.....	.....	0.92	9.5	5.0	...	....	38.0	Tetany relieved by calcium gluconate; died next day
S. M. V., 6 years‡	Extensive burn	3/15/31	Carpopedal spasm; stiff face; generalized twitching	.....	.....	0.94	12.6	5.4	...	....	....	Tetany relieved by the administration of calcium gluconate; died of septicemia
M. W., 2 years	Bronchopneumonia	4/16/33	Carpopedal spasm; Chvostek sign; rigidity	CCC. 1.1; ACC. 0.7; AOC. 1.3; COC. 1.8	Healed rickets	0.66	10.2	4.5	6.4	7.39	61.0	Tetany relieved by the administration of calcium gluconate intravenously
		4/21/33	None	CCC. >5.0; ACC. 3.8; AOC. >5.0; COC. 5.0	.....	0.43	....	...	...	....	....	Recovered

* CCC. indicates cathodal closure contraction; ACC., anodal closure contraction; AOC., anodal opening contraction; COC., cathodal opening contraction.
† Case studied and reported through the courtesy of the medical service of Vanderbilt University Hospital.
‡ Case studied and reported through the courtesy of the surgical service of Vanderbilt University Hospital.

We have already mentioned [2] this type of tetany as one symptom of the severe toxemia seen in infants with alimentary intoxication or post-influenzal pneumonia. The manifestations of tetany were, as a rule, transient and were so small a part of the general intoxication that the nature of the nervous hyperirritability was not investigated in detail. The observation was made repeatedly, however, that the blood of such infants frequently contained increased concentrations of guanidine bases with no decrease in the calcium content. Because of the well recognized antagonism [3] between the physiologic action of guanidine and calcium salts it seemed possible that the increased guanidine might be sufficient to decrease the effect of the normal amount of calcium and so cause manifestations of tetany. We therefore studied more thoroughly a few cases in which tetany was an outstanding part of the clinical picture. Data from eleven such cases are presented in the accompanying table. In these instances the concentration of calcium in the blood is normal or as high as is compatible with the observed protein content of the serum according to the chart of Peters and Eiserson.[4] In the instances in which roentgenograms of the extremities were taken there was no evidence of rickets. There were no changes in the inorganic phosphate values or the base bicarbonate of the blood sufficient to account for the tetany. The electric reactions were not tested in every case, but whenever they were obtained they were typical of tetany, and strengthened the argument that this diagnosis was correct in spite of the absence of the usual chemical changes in the blood. This contention was further supported by the relief of the hyperirritability by calcium medication. The only positive finding which offers an explanation of the tetany is the increase in guanidine in the blood which occurred in every instance.

Various points which have been brought out in experimental work in animals in regard to the toxic picture associated with hyperguanidinemia help in understanding the condition we have observed clinically. Two factors which favor the occurrence of increased guanidine in the blood

2. Minot, A. S., and Dodd, K.: The Occurrence of Increased Guanidine as a Complicating Factor in Certain Clinical Conditions in Children, Am. J. Dis. Child. **46**:522 (Sept.) 1933.

3. (*a*) Kühnau, J., and Nothmann, M.: Ueber die Guanidintoxikose und ihre Beziehungen zur Tetanie, Ztschr. f. d. ges. exper. Med. **44**:505, 1925. (*b*) Clark, G. A.: Interrelation of Parathyroids, Suprarenals, and Pancreas, J. Physiol. **58**: 294, 1924. (*c*) Minot, A. S., and Cutler, J. T.: Guanidine Retention and Calcium Reserve as Antagonistic Factors in Carbon Tetrachloride and Chloroform Poisoning, J. Clin. Investigation **6**:369, 1928.

4. Peters, J. P., and Eiserson, L.: The Influence of Protein and Inorganic Phosphorus on Serum Calcium, J. Biol. Chem. **84**:155, 1929.

have recently been reported.[5] They are increased autolysis of the tissues and rapid dehydration. Whether or not a moderate increase in guanidine causes symptoms of intoxication depends partly on the length of time such an increase is maintained and also on the state of calcium nutrition of the subject. The work of Minot and Cutler [3c] and, more recently, that of Bryan, Minot and Chastain [6] showed that feeding adult dogs for several weeks with a diet low in calcium induces a degree of calcium deficiency which, although insufficient to be recognized by any objective symptoms or by a reduction in the calcium content of the blood, greatly increases the susceptibility of the animals to the toxic action of guanidine. A prolonged maintenance of increased amounts of guanidine in the blood will, however, cause symptoms of intoxication even in an animal on a diet adequate in calcium.

The manifestations of guanidine intoxication vary considerably in different animals, even when experimental poisoning is produced directly by the administration of guanidine hydrochloride. These differences are even more marked when the intoxication is induced by causing extensive destruction of the tissue or rapid dehydration. In severe intoxication which develops rapidly nervous hyperexcitability may be transient or entirely absent, and the picture may be one of extreme depression. When the hyperguanidinemia is more moderate and prolonged the manifestations of tetany are more common. Occasionally tetany is the single outspoken symptom. More often it occurs with other manifestations of the general intoxication as well as symptoms referable to the primary condition which has caused the hyperguanidinemia. When the tetany is outspoken, we have observed, as have other investigators,[7] that the same changes in electric excitability accompany the nervous hyperirritability induced by guanidine as are seen in tetany caused by hypocalcemia.

The clearest example of tetany induced by guanidine in the absence of other symptoms to complicate the picture is afforded by experiments reported by Bryan, Minot and Chastain.[6] When the guanidine increases

5. Bryan, W. R.; Minot, A. S., and Chastain, L. L.: Hyperguanidinemia Associated with Dehydration in Normal and Parathyroidectomized Dogs, Am. J. Physiol. **106**:738, 1933. Minot and Dodd.[2]

6. Bryan, W. R.; Minot, A. S., and Chastain, L. L.: The Occurrence of Guanidine as a Contributory Factor in Parathyroid Tetany. The Influence of Guanidine on the Onset and Severity of Tetany, to be published.

7. Paton, D. N., and Findlay, L.: Tetania Parathyreopriva: Its Nature, Cause, and Relations to Idiopathic Tetany: III. Changes in Peripheral Nerves and Muscles, Quart. J. Exper. Physiol. **10**:243, 1916. Frank, E., Stern, R., and Nothmann, M.: Die Guanidin- und Dimenthylguanidin-Toxikose des Säugetiers und ihre physio-pathologische Bedeutung, Ztschr. f. d. ges. exper. Med. **24**:314, 1921.

moderately in the blood as a result of dehydration in parathyroidectomized dogs, typical tetany may be precipitated before the calcium in the blood has decreased significantly. In these instances the moderate hyperguanidinemia is maintained for too short a time to produce other symptoms, and yet it is sufficient to cause hyperexcitability of the nervous system when the calcium metabolism is slightly disturbed.

In the clinical instances of tetany which we have presented the cause for the increase in guanidine is not always apparent. Sometimes there is undoubtedly increased autolysis of the tissues. Usually there is a more or less severe condition of dehydration. Often the history of the previous diet makes it certain that the state of calcium nutrition is poor in spite of the fact that the calcium of the blood is normal. The moderately reduced concentration of inorganic phosphate observed in some instances is further evidence of faulty calcium-phosphate nutrition, even though roentgenograms showed no evidence of rickets. In practically every instance the tetany was relieved by the administration of calcium. Although in these eleven cases tetany was, for a time at least, an outstanding symptom, it was usually accompanied by symptoms of the severe primary illness. This is even more true in the transient manifestations of tetany referred to earlier in the paper.

The clinical data presented here are inadequate to establish the contention that the tetany is certainly due to guanidine. However, there seems to be no doubt that tetany was present according to the various accepted criteria for diagnosing this condition. The changes in the inorganic constituents of the blood are insufficient to account for the tetany. Experimental work which we have cited has shown that moderate increases in guanidine may cause tetany even in the absence of a decreased blood calcium. The conclusion seems justified that these are instances of tetany due primarily to the toxic action of guanidine.

GROWTH AND BASAL METABOLISM

I. BASAL METABOLISM OF PRESCHOOL CHILDREN

ITSIRO NAKAGAWA, M.D.
TOKYO, JAPAN

Since improvement of the nutrition of children is being much stressed, it is indispensable that one should study the food requirement. There is much virgin soil for the study of the basal metabolism of children, especially of preschool children. Few investigations have been made on children of this age group because of the difficulty of securing subjects and because of the obstruction to accurate testing caused by the whimsical disposition of the children.

In this paper I shall present a general report on the basal metabolism of preschool children and the influence thereon of sex and race.

EXPERIMENTAL METHOD

Thirty-one children, fifteen boys and sixteen girls, between the ages of 3 years and 10 months and 6 years and 6 months served as subjects. All of them were healthy, and their weight, height, chest girth and other physical characteristics were about the average for Japanese children of the respective ages (table 1).

Before the experiments reported in this paper were conducted, some preparatory training was given to accustom the children to the test, lest they should entertain fear of the unfamiliar calorimeter. All the metabolism tests were made early in the morning, when the subjects were in a postabsorptive state, after a rest period of not less than thirty minutes. The children were kept quiet during the experimental period; while their general condition and activities were observed through the glass window in the respiration chamber, the attendant nurses ascertained that they were quiet by recording the respiratory rate and the pulse rate, which are instantly increased by muscular activity. A normal diet was given them in the institute in order to avoid the least influence on the metabolism by the previous diet. A diet rich in protein has a close relation with a higher basal metabolic level. The lowest figures, according to Krogh, are obtained after a diet rich in carbohydrate. All the tests were conducted in autumn (1932) with a view to excluding a seasonal influence. Benedict's respiration apparatus with a cot chamber was employed for measuring the heat production. The accuracy of the apparatus was established by conducting alcohol checks, the results of which are shown in table 2.

The results obtained were expressed in terms of total calories per hour, calories per square meter of body surface per hour, calories per kilogram of body weight per twenty-four hours and total calories per twenty-four hours (table 3).

TABLE 1.—*Physical Characteristics of the Children Tested*

No.	Name	Age Yrs.	Age Mos.	Weight, Kg.	Height, Cm.	Chest Girth, Cm.	Sitting Height, Cm.	Body Surface, Sq.M.	Pulse Rate	Respiratory Rate	Temperature, C.
Boys											
1	S. N.	3	10	13.45	94.8	55.0	50.0	0.59	86	22	36.2
2	Y. T.	4	3	14.30	96.7	54.5	53.2	0.60	82	23	36.0
3	K. T.	4	5	14.90	96.3	54.5	53.5	0.62	104	24	36.7
4	M. M.	4	5	16.45	106.9	61.5	....	0.70	97	22	36.6
5	H. K.	4	5	16.00	102.2	58.3	52.5	0.67	102	24	36.3
6	I. M.	4	7	16.00	101.8	59.3	52.0	0.67	92	..	36.2
7	E. O.	4	9	16.85	104.2	59.5	53.5	0.69	...	..	36.7
8	T. K.	5	1	15.80	102.7	59.0	54.0	0.67	88	22	35.9
9	N. M.	5	2	14.90	99.2	57.4	54.0	0.63	92	24	36.5
10	Y. T.	5	8	16.65	105.3	62.3	54.0	0.69	84	23	36.4
11	T. T.	5	8	17.30	106.0	60.6	54.4	0.71	95	26	36.9
12	T. I.	5	11	17.75	114.1	65.3	58.5	0.76	94	17	36.5
13	E. H.	5	11	18.25	110.0	62.7	56.0	0.74	86	20	36.0
14	N. M.	6	5	19.75	112.0	65.3	58.0	0.78	78	21	35.8
15	Y. T.	6	6	19.30	114.9	66.5	55.5	0.79	82	..	36.0
Girls											
16	S. N.	4	0	15.00	98.1	56.0	53.5	0.63	101	26	36.3
17	T. I.	4	10	14.45	96.7	54.2	49.5	0.61	85	21	36.7
18	T. N.	4	10	12.95	95.3	53.5	48.0	0.58	98	20	35.9
19	N. N.	5	1	14.75	99.5	56.2	51.0	0.63	104	..	36.6
20	Y. M.	5	1	16.50	103.6	59.0	53.0	0.68	92	21	36.2
21	E. S.	5	4	14.60	102.1	57.2	52.0	0.64	108	24	36.5
22	Y. F.	5	4	17.50	105.7	61.3	54.5	0.71	82	22	35.8
23	M. Y.	5	5	16.50	109.3	62.0	53.5	0.71	100	..	36.2
24	R. N.	5	6	14.95	101.8	59.3	53.0	0.65	...	21	36.2
25	R. Y.	5	7	17.05	110.8	63.7	53.0	0.73	88	26	36.5
26	M. O.	5	9	19.35	108.8	63.5	54.5	0.76	90	23	36.5
27	T. T.	5	11	15.90	105.3	62.2	53.0	0.68	100	20	36.5
28	Y. F.	6	1	16.70	109.6	63.0	52.5	0.72	98	22	36.2
29	T. K.	6	1	19.85	115.6	67.0	54.0	0.80	96	23	36.0
30	T. T.	6	5	17.60	109.8	62.5	53.5	0.73	82	18	36.6
31	E. M.	6	6	18.45	112.4	64.8	54.5	0.76	80	20	36.6

TABLE 2.—*Results of Tests of Accuracy of the Apparatus*

Date	Period	Minutes	Oxygen Consumed, Liters	Carbon Dioxide Produced, Liters	Respiratory Quotient*
7/18/32	I	30	6.906	4.585	0.664
	II	30	6.688	4.539	0.679
	Average				0.672
8/29/32	I	30	7.135	5.013	0.703
	II	30	7.702	5.013	0.651
	Average				0.677
9/19/32	I	30	5.924	3.868	0.653
	II	30	6.403	4.158	0.649
	Average				0.651
10/31/32	I	30	2.121	1.394	0.657
	II	30	2.021	1.359	0.672
	Average				0.665
11/14/32	I	30	4.347	2.819	0.648
	II	30	4.397	2.885	0.656
	Average				0.652
12/26/32	I	30	5.204	3.257	0.626
	Average				0.653
	Total average				0.662

* Standard, 0.666.

Calculation of the Surface Area of the Body.—Though Benedict and Talbot[1] insisted that the heat production of the body depends on the gross quantity of active protoplasm (mass), the unit of surface area is still in general use. There are, however, various opinions as to the formula and method for calculating the surface

TABLE 3.—*Results of Tests*

No.	Name	Oxygen Consumed, Liters	Carbon Dioxide Produced, Liters	Respiratory Quotient	Nitrogen Excreted in Urine, Gm.	Basal Heat Production: Calories per Hour: Total	Basal Heat Production: Calories per Hour: Per Sq.M.	Basal Heat Production: Calories per Day: Per Kg.	Basal Heat Production: Calories per Day: Total	Burned Material: Protein, per Cent	Burned Material: Carbohydrate, per Cent	Burned Material: Fat, per Cent
Boys												
1	S. N.	7.254	5.730	0.79	0.2380	33.98	57.59	60.63	815.52	18.0	23.5	58.5
2	Y. T.	6.860	5.782	0.84	0.2564	32.67	54.45	54.83	784.08	20.1	39.2	40.7
3	K. T.	7.832	6.768	0.86	0.3112	37.42	60.35	60.27	898.08	21.3	46.6	32.1
4	M. M.	7.846	6.728	0.86	0.2168	37.56	53.66	54.80	901.44	14.8	47.5	37.7
5	H. K.	6.840	5.538	0.81	0.1844	32.43	48.40	48.65	778.32	14.6	30.2	55.2
6	I. M.	7.416	6.168	0.83	0.3324	35.01	52.25	52.52	840.24	24.3	34.5	41.2
7	E. O.	7.992	6.860	0.86	0.2149	38.30	55.51	54.55	919.20	14.3	47.8	37.9
8	T. K.	7.100	6.300	0.89	0.1890	34.39	51.33	52.24	825.36	14.1	56.7	29.2
9	N. M.	7.262	6.004	0.83	0.2228	34.53	54.81	55.62	828.72	16.5	35.2	48.3
10	Y. T.	8.012	6.484	0.81	0.2406	37.90	54.93	54.63	909.60	16.3	29.6	54.1
11	T. T.	8.004	6.830	0.85	0.2735	38.07	53.62	52.81	913.68	18.4	45.5	36.1
12	T. I.	7.826	6.350	0.81	0.2186	37.16	48.89	50.24	891.84	15.1	30.1	54.8
13	E. H.	8.530	6.818	0.80	0.1615	40.45	54.66	53.19	970.80	10.2	28.7	61.1
14	N. M.	7.878	6.716	0.85	0.2303	37.77	48.42	45.90	906.48	15.6	44.2	40.2
15	Y. T.	7.168	6.666	0.93	0.1522	35.08	44.41	43.62	841.92	11.1	73.8	15.1
Girls												
16	S. N.	6.998	5.578	0.80	0.2027	32.88	52.19	52.61	789.12	15.8	26.9	57.3
17	T. I.	6.252	6.158	0.98	0.2265	30.72	50.36	51.02	737.28	18.9	81.1	0.5*
18	T. N.	6.096	5.476	0.90	0.2349	29.27	50.47	54.25	702.48	20.6	60.5	18.9
19	N. N.	6.928	6.352	0.92	0.2498	33.50	53.17	51.78	804.00	19.1	67.1	13.8
20	Y. M.	7.382	6.270	0.85	0.1994	35.28	51.88	51.32	846.72	14.5	44.8	40.7
21	E. S.	6.570	5.374	0.82	0.2213	31.10	48.59	51.12	746.64	18.2	31.7	50.1
22	Y. F.	7.004	6.340	0.91	0.2888	33.68	47.44	46.19	808.32	22.0	62.1	15.9
23	M. Y.	7.336	6.260	0.85	0.2386	34.90	49.15	50.76	837.60	17.5	46.0	36.5
24	R. N.	7.196	5.750	0.80	0.2027	33.91	52.17	54.44	813.84	15.3	27.1	57.6
25	R. Y.	7.520	6.554	0.87	0.1084	36.41	49.88	51.25	873.84	7.6	54.7	37.7
26	M. O.	7.910	6.138	0.78	0.2512	37.03	48.72	45.93	888.72	17.4	18.0	64.6
27	T. T.	8.246	6.514	0.79	0.1693	38.95	57.28	58.79	934.80	11.1	25.4	63.5
28	Y. F.	7.948	5.984	0.75	0.2075	37.25	51.74	53.53	894.00	14.3	9.9	75.8
29	T. K.	7.932	6.494	0.82	0.2491	37.62	47.03	45.49	902.88	17.0	32.2	50.8
30	T. T.	7.924	6.036	0.89	0.2038	37.24	51.01	50.32	893.76	14.0	12.9	73.1
31	E. M.	7.094	6.392	0.90	0.2798	34.17	44.96	44.45	820.08	21.0	60.2	18.8

* Stored as fat.

area of the body of a child. In using Meeh's formula $K \sqrt[3]{} P^2$, in which W represents the weight in kilograms, Lissauer chose $K = 10.3$ as the best value for general use with children. Faber and Melcher calculated the body surface of one hundred new-born infants according to the linear formula and suggested that for

1. Benedict, F. G., and Talbot, F. B.: Metabolism and Growth from Birth to Puberty, Washington, D. C., Carnegie Institution, 1921, publ. 302. Talbot, F. B.: Standards of Basal Metabolism in Normal Infants and Children, Am. J. Dis. Child. **21**:519 (June) 1921.

infants the Du Bois [2] height-weight formula be modified and a constant of 78.5 used instead of 71.84. The measurements of Pfaundler and Woerner indicate that the height-weight formula may be applied to children with accuracy. Benedict and Talbot gave eight constants, according to the body weight and the sex of children, as against Meeh's formula. In Japan, Ohtani [3] is the only one who has measured the body surface of children. But his results were based on undernourished children, and he was uncertain of the measurements for girls. So the method and formula for determining the body surface of children are rather obscure. I wish in the near future to have a chance to measure the body surface of children. In these studies the Du Bois height-weight formula was used for convenience.

RESULTS

Influence of Sex on Basal Metabolism.—Benedict stated that the reason for the discrepancy in heat production between boys and girls is that girls have less active tissue than boys but much fat tissue. The

TABLE 4.—*Percentage Difference in Basal Heat Production Between Boys and Girls*

	Per Sq. Meter per Hour			Per Kg. per Day			Per Day		
	Calories		Deviation, per Cent	Calories		Deviation, per Cent	Calories		Deviation, per Cent
Age	Boys	Girls		Boys	Girls		Boys	Girls	
4	54.89	52.19	4.9	55.84	52.61	5.8	835.49	789.12	5.6
5	53.48	50.40	5.8	53.73	51.36	4.4	853.38	787.11	7.8
6	50.82	50.09	1.4	50.07	49.97	0.0	905.72	886.87	2.1

difference in heat production in the two sexes is apparent in my results (table 4), but the percentage of difference is not so great as for adults.

Influence of Age on Basal Metabolism.—That the heat production of children per unit of body weight and body surface is much higher than that of adults and decreases progressively during the period of growth is well known. In my results, the change of heat production with age is observable in so short a period as three years (table 5).

Comparison with Foreign Children.—During recent years much interest has been centered on the study of racial influence on basal metabolism, especially in adults. While one author recognizes a racial influence, another denies its existence. Takahira [4] said that the heat production of the Japanese is essentially the same as that of the American and the European. Krishman and Vareed [5] studied the basal

2. Du Bois, E. F.: Basal Metabolism in Health and Disease, Philadelphia, Lea & Febiger, 1927, p. 165.

3. Ohtani, K.: Measurement of Body Surface of Japanese Children, Tokyo Ijishinshi, December, 1907.

4. Takahira, H.: The Basal Metabolism of Normal Japanese Men and Women, Rep. Imp. Gov. Inst. for Nutrition **1**:1 (Jan.) 1925.

5. Krishman, B. T., and Vareed, C.: Basal Metabolism of Young College Students in Madras, Indian J. M. Research **19**:831, 1932.

TABLE 5.—*Variations in Basal Heat Production and in Physical Characteristics According to Age* *

Age, Years	Number of Subjects	Weight, Kg.	Height, Cm.	Chest Girth, Cm.	Sitting Height, Cm.	Body Surface, Sq.M.	Pulse Rate	Respiratory Rate	Temperature, C.	Oxygen Consumed, Liters	Carbon Dioxide Produced, Liters	Respiratory Quotient	Nitrogen Excreted in Urine, Gm.	Basal Heat Production: Calories per Hour per Sq.M.	Basal Heat Production: Calories per Day: Per Kg.	Basal Heat Production: Calories per Day: Total	Burned Material: Protein, per Cent	Burned Material: Carbohydrate, per Cent	Burned Material: Fat, per Cent
Boys																			
4	5	15.02	99.4	56.8	52.3	0.64	94	23	36.4	7.327	6.109	0.83	0.2414	54.89	55.84	835.49	17.8	37.4	44.8
5	4	15.89	102.0	58.8	53.4	0.67	91	23	36.3	7.443	6.333	0.85	0.2398	53.48	53.73	853.38	17.3	43.6	39.2
6	6	18.17	110.4	63.8	55.2	0.75	87	21	36.3	7.903	6.644	0.84	0.2128	50.82	50.07	905.72	14.5	42.0	43.6
Girls																			
4	1	15.00	98.1	56.0	53.5	0.63	101	26	36.3	6.998	5.578	0.80	0.2027	52.19	52.61	789.12	15.8	62.9	57.3
5	8	15.28	101.8	57.8	51.8	0.65	96	22	36.4	6.846	5.998	0.88	0.2328	50.40	51.36	787.11	18.3	52.6	33.4
6	7	17.84	110.3	63.8	53.6	0.74	91	22	36.4	7.796	6.302	0.83	0.2099	50.09	49.97	886.87	14.6	30.5	55.6

* Age is taken at the nearest birthday; e. g., a child is considered 4 years old at any time between 3½ and 4½ years.

metabolism of seventy-six college students, sixty-one men and fifteen women, in Madras, India, and compared the results with the Du Bois standards. They found a deviation of —12 per cent from the Du Bois standards but stated that this was due not to a racial influence but to the easy relaxation of muscles in a tropical climate and to the lack of protein in the average diet in Madras.

TABLE 6.—*Basal Heat Production per Day and Percentage of Deviation from Benedict-Talbot Standards*

No.	Name	Age Yrs.	Age Mos.	Observed Value, Calories	Percentage Deviation (Increase) from Standards Referred to Weight
Boys					
1	S. N.	3	10	816	20.4
2	Y. T.	4	3	784	11.4
3	K. T.	4	5	898	24.4
4	M. M.	4	5	901	17.3
5	H. K.	4	5	778	3.0
6	I. M.	4	7	840	11.3
7	E. O.	4	9	919	18.1
8	T. K.	5	1	825	10.2
9	N. M.	5	2	829	14.8
10	Y. T.	5	8	910	17.7
11	T. T.	5	8	914	16.0
12	T. I.	5	11	892	11.5
13	E. H.	5	11	971	7.1
14	N. M.	6	5	906	6.1
15	Y. T.	6	6	842	0.0
Average				868	12.6
Girls					
16	S. N.	4	0	789	14.3
17	T. I.	4	10	737	8.7
18	T. N.	4	10	702	9.7
19	N. N.	5	1	804	17.4
20	Y. M.	5	1	847	17.3
21	E. S.	5	4	747	9.9
22	Y. F.	5	4	808	8.2
23	M. Y.	5	5	838	16.1
24	R. N.	5	6	814	18.0
25	R. Y.	5	7	874	18.6
26	M. O.	5	9	889	12.5
27	T. T.	5	11	935	32.1
28	Y. F.	6	1	894	23.0
29	T. K.	6	1	903	12.5
30	T. T.	6	5	894	19.2
31	E. M.	6	6	820	6.5
Average				831	15.3

Results Obtained by Wang and Hawk			
Number of Subjects	Age, Years	Observed Value, Calories	Percentage of Deviation from Standards Referred to Weight
11	5 to 11		
Average		1,049	16.6
9	5 to 14		
Average		1,024	18.5

There are few reports, however, comparing the heat production of oriental with that of occidental children. Wang and Hawks [6] studied the basal metabolism of twenty-one Chinese children reared in the United States and reported that all of them had a total heat production above the Benedict-Talbot standards, with an average percentage of deviation ranging from +6.8 to +16.6 for the boys and from +11.5 to

6. Wang, C. C., and Hawks, J. E.: Basal Metabolism of Twenty-One Chinese Children Reared or Born and Reared in the United States, Am. J. Dis. Child. **44:** 69 (July) 1932.

+ 20.2 for the girls. A comparison of the results obtained in my tests with the Benedict-Talbot standards is presented in table 6.

I hope to make a further study to determine whether or not this deviation is the result of racial difference.

CONCLUSION

The basal heat production of thirty-one preschool children, fifteen boys and sixteen girls, between the ages of 3 years and 10 months and 6 years and 6 months has been studied.

The basal heat production per twenty-four hours increases with age, as do weight, height, sitting height, chest girth and other physical measurements. But the basal heat production per unit of body weight and of surface area decreases with age, as do the pulse rate and the respiratory rate.

My tests showed a difference in heat production between boys and girls. However, the percentage of difference is not so great as that between the two sexes in adult life.

All the children tested had a total heat production above the Benedict-Talbot standards, and the percentage of deviation agrees with that noted for oriental children by Wang and Hawks in the United States.

OBSTRUCTIVE LARYNGITIS

A CRITICAL ANALYSIS OF THREE HUNDRED AND FIFTY-TWO CASES

ERLING S. PLATOU, M.D.
AND
HERMAN HILLEBOE, M.D.
MINNEAPOLIS

Obstructive laryngeal dyspnea, a serious symptom complex which often requires emergency relief, develops so frequently from an apparently innocent case of laryngitis, that it remains a constant problem to general practitioners and pediatricians.

Although the causes of this condition are numerous, it is our intention to limit this paper to a consideration of cases of infectious origin. The appalling institutional mortality from obstructive laryngitis, often due to delay in the recognition of impending asphyxia and its causes, demands greater diligence for early precise differentiation in that group of conditions commonly designated as "croup." During the past twelve years, 352 patients with alleged laryngeal diphtheria were admitted to the contagious division of the Minneapolis General Hospital. Of this number 103 patients, or 29 per cent, died—a mortality rate which should emphasize the seriousness of this type of disease. Thirty-five of the deaths occurred during or very shortly after admission, in spite of relief from the obstruction, indicating that a large number of patients receive too much expectant therapy before hospitalization. Since no opportunity was afforded for adequate treatment in these cases they are excluded in the consideration of the effectiveness of various methods of treatment. The present study is based, therefore, on observation in 317 cases, of which 255 were cases of diphtheritic and 62 cases of non-diphtheritic laryngitis. The 255 cases of diphtheritic laryngitis will be discussed first. This group can be classified and defined conveniently as follows:

1. *Minimal.*—This class comprises 117 patients with obstructive laryngeal symptoms, positive bacteriologic evidence of diphtheria, edema and sometimes a small amount of thin adherent membrane in the larynx only (table 1 and fig. 2).

2. *Moderately Advanced.*—Patients with symptoms of obstructive laryngitis, usually positive bacteriologic evidence of diphtheria and considerable amounts of

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From the Department of Pediatrics, University of Minnesota and the Contagious Division of the Minneapolis General Hospital.

fairly loose membrane in the larynx, but with little or none in the pharynx or trachea, make up this group of 52 (table 1 and fig. 2).

3. *Advanced.*—Included here are the patients having laryngeal symptoms and large amounts of pharyngeal membrane, usually with positive bacteriologic evidence of diphtheria. Those with a tracheobronchial membrane are also placed in this category. There were 69 with the former and 17 with the latter type of case (table 1 and fig. 2).

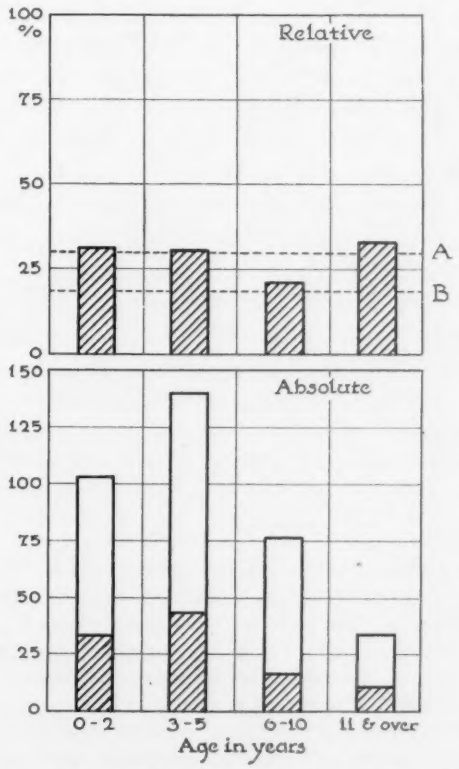

Fig. 1.—Incidence and mortality in diphtheritic and nondiphtheritic cases: *A*, average mortality for 352 cases; *B*, average mortality for nonmoribund cases (in 352 cases).

Direct laryngoscopy has been done in practically all cases of obstructive laryngitis since 1925. In the more urgent cases, it was done at once. The procedure enables the operator to relieve dyspnea temporarily by inserting a metal catheter into the larynx, until the most suitable means for permanent relief can be decided on. In the absence of emergency, laryngoscopy was deferred until a painstaking history, careful physical examination, roentgen examination of the chest, and laboratory tests could be completed. Unless rendered unnecessary by the information gained in this manner, direct inspection of the larynx was then per-

formed for the purpose of determining the cause of the obstruction. Use of the laryngoscope permitted visual diagnosis as well as the opportunity of taking smears and cultures. Smears stained with Gram's, Neisser's, methylthionine chloride, U. S. P. (methylene blue), and Ziehl's stain were examined immediately to determine the type of causative organism present. Cultures on Löffler's and blood agar mediums were then taken to confirm the immediate diagnosis.

When the cause of obstructive laryngitis was determined and signs of air hunger appeared, such as suprasternal epigastric and intercostal retraction, restlessness, anxiety, refusal of fluids and increasing pulse rate, prompt surgical intervention was resorted to before cyanosis appeared. The ideal method of operation in given types of cases has been a subject of considerable controversy in the past. With a desire to arrive at some conclusion in regard to this question, the present study was undertaken.

Inspection of table 1 and figure 2 reveals that of the 255 nonmoribund, treated patients, 43 or 17 per cent died. It would appear that the mortality was proportional to the severity of the cases, the percentage ranging from 8.5 in the minimal class and 15 in the moderately advanced to 29 in the advanced class. In order to substantiate the validity of our clinical impressions regarding differences in percentage, we have employed standard statistical methods.

STATISTICAL METHOD

In small series of cases chance alone can be the cause of apparent differences between percentages. Modern statistical technic makes it possible to determine how often chance would give specific differences in a given series. If chance is the cause of a difference only 5 times in 100, the statistician commonly assumes that the particular difference is not 1 of those 5; hence the difference between percentages is probably not due to chance and is a real one. For the sake of brevity, it is usually said that the difference is significant; if the possibilities are less than 5 in 100, that it is due to chance alone. In statistics this factor of chance is obtained by determining the probability integral, which when multiplied by 100 gives the "chances in 100."

The application of these methods to our data includes the following technic:[1]

p = proportion of cases, i. e., $\frac{\text{(number dying)}}{\text{(total number of cases)}}$

$q = 1 - p$

n = number of cases

Standard deviation of proportion $p = \sqrt{\frac{p\,q}{n}}$

The proportion p is essentially the same as percentage, except that it is left in terms of 1, or unity, instead of 100, or per cent. The standard deviation is a statistical constant which indicates that two thirds of the cases in similar series

1. Dunn, H. L.: Physiol. Rev. **9**:275, 1929.

of the same types of cases will fall between the amount of the standard deviation added to or substracted from the proportion, p. If $p = 0.25$ and the standard deviation of $p = 0.5$, two thirds of the cases would fall between $(0.25 + 0.5) = 30$ and $(0.25 - 0.5) = 20$. To test the significant difference between two proportions (p's) we need one more constant, and that is the standard error of the difference itself. The formula for this constant is $\sigma(p_1 - p_2) = \sqrt{\sigma p^2 + \sigma p_2^2}$. When the difference between the two proportions is 2 times its standard error $[(p_1 - p_2) = 2(\sqrt{\sigma p_1^2 + \sigma p_2^2})]$, the difference is assumed to be significant since it would occur only 5 times in 100 due to chance. When the difference is 3 times its standard error, there is less than 1 chance in 100 that the difference is caused by chance alone.

For example, let us test the difference in percentage mortality in the moderately advanced (15 per cent) and the advanced (29 per cent) classes. Would this difference in percentages be caused by chance?

$$\text{p moderately advanced} = \frac{8\ \text{(dead)}}{52\ \text{(no. cases)}} = 0.154\ (0.154 \times 100\ 15.4\%)\ \sigma\text{p moderately advanced} = \sqrt{\frac{p\ q}{n}} = \sqrt{\frac{0.154 \times 0.946}{52}} = 0.050.$$

$$\text{p advanced} = \frac{25\ \text{(dead)}}{86\ \text{(no. cases)}} = 0.291\ (0.291 \times 100) = (29.1\%)\ \sigma\text{p advanced} = \sqrt{\frac{p\ q}{n}} = \sqrt{\frac{291 \times 0.719}{86}} = 0.049.$$

The difference in proportions $= 0.291 - 0.154 = 0.137$. The standard error of the difference $= \sigma(\text{pn-a-pa}) = \sqrt{\sigma\text{pna}^2 + \sigma\text{pa}^2} = \sqrt{(0.05)^2 + (0.049)^2} = 0.070$. The difference (0.137) is practically 2 times its standard error (0.070); therefore the difference is probably significant, because it would be due to chance only 5 times in 100. Similarly there is a significant difference in the proportion dying in the minimal class (8.5 per cent) and in the advanced (29 per cent), less than 1 in 1,000 due to chance. However, the difference in percentage mortality between the minimal (8.5 per cent) and the moderately advanced class (15.4 per cent) could be caused by chance 22 times in 100, other factors being equal.

Before comparing the percentage mortality for different classes, it will be necessary to determine that the number of cases in each class has a similar age distribution. For example, if most of the patients are under 1 year of age in one class and over 10 years of age in another class, this difference must be taken into consideration. In our data, the number of cases in each age group in the minimal class and in the moderately advanced class does not differ significantly, showing that the cases are distributed similarly with respect to age and number and that the groups, therefore, are comparable. The moderately advanced and advanced classes do not differ from each other. However, the minimal and advanced classes do differ; this difference is probably due to the fact that in the advanced class a greater number of patients are over 6 years of age (table 1). Consequently, in any subsequent comparison of the minimal and advanced classes, we must consider this difference in age distribution.

The distribution of cases with respect to age, sex, type and number in each group has been tested by the technic described.

MATERIAL

Table 1 reveals that 68 per cent of the patients with diphtheritic laryngitis were under 5 years of age; 26 per cent of the total were

under 2 years of age. The group from 3 to 5 years of age made up the greatest number of cases (42 per cent). The most common age of occurrence of obstructive laryngitis due to diphtheria is, therefore, from 3 to 5 years inclusive. The next in order of frequency is from birth to 2 years, from 6 to 10 years, and last, over 10 years.

Regarding sex distribution in our series, there were significantly more males (61 per cent) than females (39 per cent), table 1 and figure 2. This was uniformly constant when we compared the relative number of males in the three classes.

The largest number of cases were of the minimal laryngeal type (46 per cent), and the smallest number were tracheobronchial (7 per cent). The reason for the apparently large number of advanced cases in comparison to the smaller number of moderately advanced is that

TABLE 1.—*Incidence and Mortality by Age and Class in Cases of Diphtheria*

	0-2 Yrs.		3-5 Yrs.		6-10 Yrs.		11 Yrs.-Over		Total No. of	Total No. of	
Class	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Per Cent
Minimal.....	42 (22M) (20F)	16% (5M) (2F)	42 (27M) (15F)	4% (1M) (1F)	28 (18M) (10F)	3% (1M)	5 (2M) (3F)	0	117 (69M) (48F)	10 (7M) (3F)	8.5
Moderately advanced	11 (7M) (4F)	27% (1M) (2F)	24 (18M) (6F)	2% (4M) (1F)	12 (6M) (6F)	0	5 (1M) (4F)	0	53 (33M) (20F)	8 (5M) (3F)	15.0
Advanced	14 (8M) (6F)	21% (1M) (2F)	41 (29M) (12F)	31% (6M) (7F)	18 (11M) (7F)	16% (2M) (1F)	13 (6M) (7F)	46% (2M) (4F)	86 (54M) (32F)	25 (11M) (14F)	29.0
Total.....	67 (37M) (30F)	19% (7M) (6F)	107 (74M) (33F)	19% (11M) (9F)	58 (35M) (23F)	6% (3M) (1F)	23 (9M) (14F)	26% (2M) (4F)	255 (155M) (100F)	43 (23M) (20F)	17.0

a large number of neglected pharyngeal cases with laryngeal involvement are included in this group. Only 17 were definite examples of tracheobronchial diphtheria.

From a study of the percentage mortality of all cases in a city institution, we are able to prognosticate the mortality of 1 person, according to age, sex and type of case. Nonmoribund infants and children up to 5 years of age inclusive show a mortality of 19 per cent. Children from 6 to 10 years of age show a rate of only 6 per cent, while in those over 10 years it is 26 per cent.

There is no significant difference in the death rate between the total number of males (15 per cent) and the total number of females (20 per cent).

Regarding complications, there were too few to warrant statistical correlation with the type of treatment used. There were 15 cases of cardiac complications, 13 of bronchopneumonia, 10 of otitis media, 7 of acute nephritis, 7 of cervical adenitis, 6 of nerve paralysis, 1 of septicemia and 1 in which there was erosion of an artery following

tracheotomy. As can be seen from the data, the number of each type of complication is too small to justify any conclusions. We were unable to prove, because of the small size of our group, the effect of any particular type of treatment on the number of complications following it. Unfortunately, we are also unable to show the important relationship between the duration of symptoms and time and type of treatment,

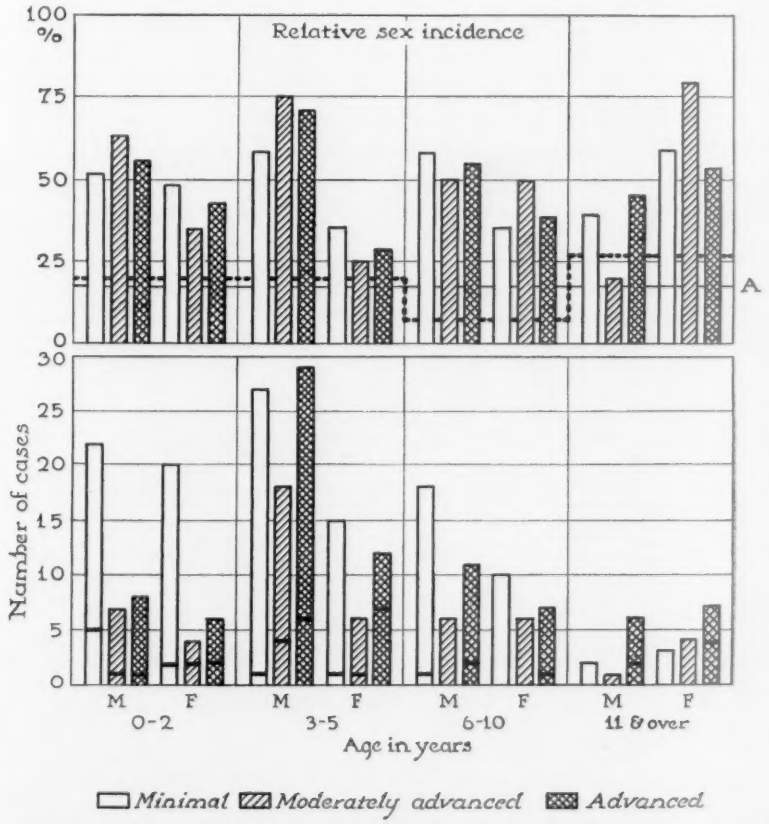

Fig. 2.—Incidence and mortality in diphtheritic cases. In the upper portion of the diagram, line *A* indicates the mortality (in per cent) for the entire group; the dotted line, the mortality for the various age groups.

because of the lack of specific data regarding the patients before they entered the hospital.

With this description of our material in mind, regarding age, sex, type and number of cases, we now consider the surgical treatment.

DIPHTHERITIC SERIES

It would seem reasonable, from a knowledge of the pathogenesis in different forms of laryngitis, that no one method of intervention will be

best suited for all cases. In fact, different stages of the same disease may present varying problems in the mechanics of obstruction. For example, edema may be the obstructive factor in early cases, whereas a membrane becomes of more importance as the disease progresses. Theoretically, the causative organism and the nature, location and extent of the membrane or of the edema should have a bearing on the selection of this method.

In all cases reported in this study the patients were given antitoxin after first being tested for serum sensitization. The antitoxin was given intravenously in all cases, except when the fear of a reaction sufficient to necessitate intervention justified intraperitoneal administration. When a diagnosis of diphtheria was established, oxygen therapy was instituted, with strict avoidance of narcotics. When definite signs of air hunger developed, operative measures were promptly undertaken.

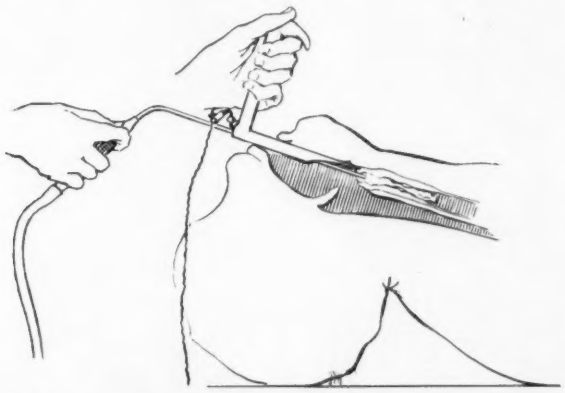

Fig. 3.—Metal catheter inserted into the larynx with the aid of a laryngoscope to accomplish laryngeal suction.

During the past few years, laryngeal suction has been applied in selected cases by means of a metal catheter inserted into the larynx with the aid of a laryngoscope, as suggested by Gover and Hardman.[2] However, many of the patients included in this study were treated prior to the introduction of this procedure, when intubation was the principal method used. We have tried to evaluate the relative merits of the different surgical methods in each type of laryngeal diphtheria by an analysis of the data previously described in 255 cases.

Treatment.—In the 117 minimal cases (table 2), 10 of the patients, 9 per cent, died. Sixty-five of these patients received medical treatment only, 52 were operated on and 9 died (17 per cent). In the 52 moderately advanced cases, 51 patients were operated on, and of these 7 died

2. Gover, R. W., and Hardman, R. P.: Arch. Pediat. **40**:170, 1923.

(14 per cent). In the 86 advanced cases, 68 patients received antitoxin only and 13 died (19 per cent). All of these patients had very mild laryngeal symptoms with neglected pharyngeal diphtheria. Of the 18 remaining patients, 12 died (67 per cent). There were, therefore, 134 patients who were not operated on, 68 of whom had predominantly a pharyngeal infection and 15 of whom died (11 per cent). In 13 of these cases death was probably due to toxemia. Of 121 patients operated on, 28, or 23 per cent died.

TABLE 2.—*Fatalities in Diphtheritic Cases*

Series	Total No.	Deaths	Per Cent	No Operation	Per Cent of Total	Deaths	Mortality, per Cent	Operation	Per Cent of Total	Deaths	Mortality, per Cent
Minimal.....	117	10	9	65	56	1	2	52	44	9	17
Moderately advanced.....	52	8	15	1*	2	1	100	51	98	7	14
Advanced.....	86	25	29	68	79	13	19	18	21	12	67
Total.....	255	43	17	134	53	15	11	121	47	28	23

* One case of laryngeal diphtheria recognized at autopsy in association with measles and bronchopneumonia.

TABLE 3.—*Fatalities in Cases in Which Intubation Was Done (Platou)*

Series	Total Number	Intubation		Intubation and Suction		Intubation, Suction and Tracheotomy		Intubation and Tracheotomy		Number Receiving Surgical Treatment after Intubation	Per Cent	Total Number of Deaths	Per Cent
		Total	Deaths	Total	Deaths	Total	Deaths	Total	Deaths				
Minimal.....	46	44	6	0	0	0	0	2	1	2	4	7	15
Moderately advanced....	28	18	2	1	0	4	2	5	2	10	36	6	21
Advanced.....	17	13	8	1	1	1	1	2	2	4	24	12	71
Total.....	91	75	16	2	1	5	3	9	5	16	18	25	27

Intubation was done in 46 minimal cases. Table 3 shows that 44 of these patients received intubation only and 6 died. Two other patients who received intubation subsequently required tracheotomy and 1 died. In 1 minimal case tracheotomy only was done and the patient died. Only 4 per cent of the minimal cases in which intubation was done required further surgical treatment, and the mortality for the entire group was only 15 per cent.

In moderately advanced cases, 28 patients were intubated (table 3). Of these, 18 received intubation only and 2 died, 10 additional patients who received intubation required further treatment, and 4 of these died. The percentage mortality for all patients in this group who received intubation was 21. One patient, who lived, was tracheotomized only. In 17 tracheobronchial cases, 13 patients were intubated only. Four of

these received subsequent tracheotomy, and 8 of the group died (62 per cent). Of the other 4 patients with tracheobronchial involvement, 1 who received intubation and suction died. One was subjected in turn to aspiration, intubation and finally tracheotomy, but died in spite of these measures. Two others were treated by intubation and tracheotomy, but died. All 4 patients in the advanced group who received intubation and further intervention died. The mortality for the entire advanced group, who were intubated without suction was 71 per cent (table 3).

Ninety-one of the entire group of 255 patients of all types were intubated. Seventy-five had intubation only, and 16 patients, or 18 per cent, received further intervention. It is significant statistically that only 4 per cent of the minimal cases in which intubation was done required additional surgical intervention, whereas 36 per cent of the

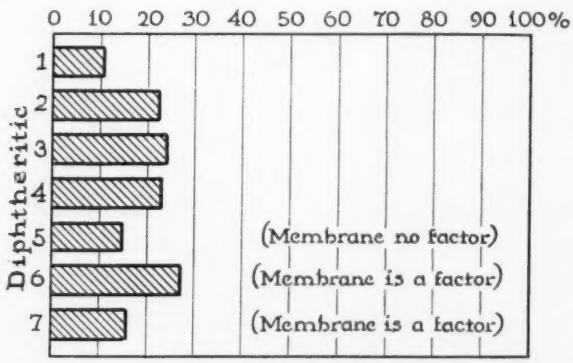

Fig 4.—Mortality among patients receiving surgical treatment: *1*, the total number of cases of Platou's series in which there was no surgical intervention; *2*, total number of Platou's series in which there was surgical intervention; *3*, 484 reported cases; *4*, 121 of Platou's cases; *5*, Platou's cases (minimal) in which intubation was done; *6*, aggregate cases, moderately advanced and advanced, in which intubation was done; *7*, aggregate cases, moderately advanced and advanced, in which suction was used.

moderately advanced cases required it. In the patients who were intubated, the mortality was 40 per cent for the two advanced classes, as compared with 15 per cent for the minimal class. These facts suggest the probability that a membrane lodged below the tube may have been responsible for death in a number of the more advanced cases.

Table 4 shows the data on all cases in our series in which laryngeal suction was used. Early in our experience, laryngeal suction was tried in 5 minimal cases, 1 of which required further surgical intervention. The other patients escaped further surgical treatment, but did not improve. Although the group is entirely too small to justify definite conclusions, it was our impression that laryngeal instrumentation aggra-

vated the dyspnea in these cases, so that its use was discontinued. Of the patients in the moderately advanced class, 27 were aspirated; in 22 this was the only procedure used. One of these died. Of the patients requiring further treatment, 2 died. In the moderately advanced class, 3 of the total number who received suction died, a mortality of only 11 per cent. Of the 3 patients in the advanced class who were treated by suction, 2, or 67 per cent, died.

Combining all types of diphtheritic laryngitis, suction was used in 35 instances (table 3). Seven patients, or 20 per cent, were later intubated, and 5 of these were secondarily tracheotomized. One patient died suddenly on the twenty-second day from profuse hemorrhage at the site of the tracheotomy wound. Of all the patients treated by suction, 8, or 23 per cent, required further intervention. Only 6, or 17 per cent, of the total series died. Although the number of patients

TABLE 4.—*Fatalities in Cases in Which Suction Was Done (Platou)*

Series	Total Number	Suction Only		Suction and Intubation		Suction, Intubation and Tracheotomy		Suction and Tracheotomy		Number Receiving Surgical Treatment after Suction		Total Number of Deaths	
		Total	Deaths	Total	Deaths	Total	Deaths	Total	Deaths		Per Cent		Per Cent
Minimal	5	4	0	0	0	0	0	1	1	1	20	1	20
Moderately advanced	27	22	1	1	0	4	2	0	0	5	19	3	11
Advanced	3	1	0	1	1	1	1	0	0	2	67	2	67
Total	35	27	1	2	1	5	3	1	1	8	23	6	17

receiving suction in our series is small, it appears that aspiration is more efficacious than intubation as a first method of relief in diphtheritic laryngitis, in which a membrane is the predominating obstructive factor. In the moderately advanced cases in which aspiration was performed, the mortality was only 11 per cent, whereas in similar cases in which intubation was used as a first means of intervention, the percentage of mortality was 21. Intubation seemed advisable, however, when edema was the chief cause of dyspnea. The mortality in patients of the last mentioned type was only 15 per cent.

In an attempt to study these facts on a larger scale, we combined our series with those of Adler [3] and of Tolle.[4] Their cases are similar to ours in age distribution, but we must assume, because of insufficient data, that the types of cases and the number of each type in their series have a similar distribution. It must be noted that Adler's cases were

3. Adler, S. W.: Minnesota Med. **9**:603, 1926.

4. Tolle, D.: Croup: An Analysis of 344 Cases, Am. J. Dis. Child. **39**:954 (May) 1930.

equally distributed as to sex, whereas Tolle's and our own cases were predominantly in males. However, since the percentage mortality is the same in males as in females, this variation in Adler's data is of no consequence in a consideration of the results of treatment.

In a study of the aggregate fatalities among 772 patients with diphtheritic laryngitis (table 5) it was found that 288, or 37 per cent, received medical treatment only; 14 of these died (5 per cent). Thirteen of the deaths, however, resulted from neglected pharyngeal diphtheria with only mild laryngeal symptoms. All cases with symptoms of laryn-

Fig. 5.—Comparison of end-results of treatment with suction and intubation: *1*, Adler's series (1920-1922); *2*, Tolle and Platou's series (1926-1932); *3a*, Platou's 28 moderately advanced cases in which intubation was done; *b*, aggregate cases, moderately advanced and advanced, in which intubation was done; *4a*, Platou's 27 moderately advanced cases in which suction was employed; *b*, aggregate cases, moderately advanced and advanced, in which suction was used; *5*, 270 cases in which treatment included suction; *6*, 772 cases in which suction was included; *7*, nondiphtheritic cases in which tracheotomy was done; *8*, nondiphtheritic cases in which suction and intubation were employed.

gitis were included in our series. In the total series of 484 patients who were operated on, 115 died, a percentage mortality of 23.8.

In our earlier cases and in Adler's series, shown in table 6, 295 were intubated as a first means of operative relief. Whereas only 23, or 8

per cent, received further operative intervention, 85 of the total number died, a percentage mortality of 29.

Of Tolle's and Platou's 194 cases (table 7) in which suction was used as a first means of operative relief, 50, or only 25 per cent, needed further intervention. Thirty-two of the entire series of patients died, a

TABLE 5.—*Aggregate Fatalities in Diphtheritic Cases*

Series	Total	Deaths	Per Cent	No Surgical Intervention	Per Cent Total	Deaths	Mortality, per Cent	Surgical Intervention	Per Cent Total	Deaths	Mortality, per Cent
Tolle.....	212	27	13	53	25	0	0	159	75	27	17
Platou.....	255	43	17	134*	52	15*	11	121	48	28	23
Adler.....	305	60	20	101	33	0	0	204	67	60	29
	772	130	17	288	37	15	5	484	63	115	24

* One patient died on the twenty-second day of complications.

TABLE 6.—*Aggregate Fatalities in Cases in Which Intubation Was Done*

Series	Total Number	Intubation Only		Intubation and Suction		Intubation Suction and Tracheotomy		Intubation and Tracheotomy		No. Receiving Surgical Treatment after Intubation		Per Cent	Total Number of Deaths	Per Cent
		Total	Deaths	Total	Deaths	Total	Deaths	Total	Deaths	Total	Deaths			
Platou.....	91	75	16	2	1	5	3	9	5	16	9	17	25	28
Adler.....	204	195	51	..	..	..	..	9	..	9	9	4	60	29
Total.....	295	270	69	2	1	5	3	18	5	23	17	8	85	29

TABLE 7.—*Aggregate Fatalities in Cases in Which Suction Was Done*

Series	Total Number	Suction Only		Suction and Intubation		Suction, Intubation and Tracheotomy		Suction and Tracheotomy		Number Receiving Surgical Treatment after Suction	Per Cent	Total Number of Deaths	Per Cent
		Total	Deaths	Total	Deaths	Total	Deaths	Total	Deaths				
Tolle.....	159	117	20	41	7	1	0	0	0	42	26	27	17
Platou.....	35	27	1	2	1	5	3	1	1	8	23	6	17
Total.....	194	144	21	43	8	6	3	1	1	50	25	33	17

mortality of 16 per cent. There was, therefore, a significant difference between the percentage mortality when suction rather than intubation was used as the initial procedure for the relief of dyspnea in selected cases of diphtheritic laryngitis.

That preventive measures have a striking effect on the incidence of diphtheria is clearly shown in figure 5. The number of cases in the city of Minneapolis has

decreased from over 1,750 a year to nearly 250 a year during the past decade. Laryngeal diphtheria has decreased in similar proportion during the same period of time, whereas the incidence of nondiphtheritic laryngitis has remained relatively constant.

NONDIPHTHERITIC SERIES

Sixty-two of the 352 cases of obstructive laryngitis in our series were nondiphtheritic in origin. These cases have been grouped according to the following classification:

1. *Acute Nondiphtheritic Laryngitis.*—Cases with obstructive laryngeal symptoms, but lacking a history of gradual progression and hoarseness, make up this group. They are characterized by an accumulation of mucus in the throat, redness and edema of the larynx and absence

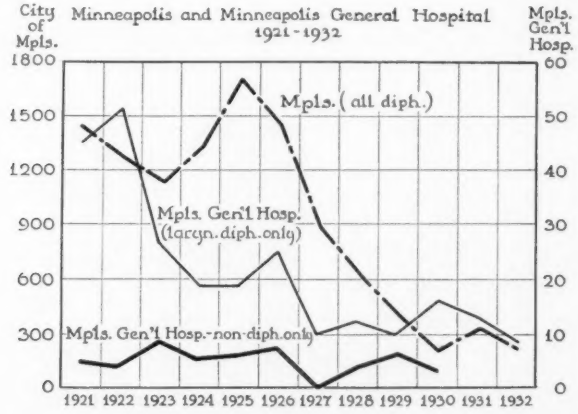

Fig. 6.—Incidence of diphtheritic and nondiphtheritic laryngitis in Minneapolis (*Mpls.*) and the Minneapolis General Hospital (*Mpls. Gen'l Hosp.*) from 1921 to 1932, inclusive.

of laryngoscopic or bacteriologic evidence of diphtheria. They are often associated with simple rhinopharyngitis. A synonym for the term suggested is acute catarrhal laryngitis.

2. *Acute Exudative Laryngotracheitis.*—Cases with obstructive laryngeal symptoms accompanied by rather profound constitutional symptoms and characterized by redness, edema, purulent or fibrinous exudate and "moss agate" plugs below the cords, but with no evidence of diphtheria make up this group. This form is not infrequently associated with influenza,[5] measles and scarlet fever.

3. *Miscellaneous Group.*—In this group are included such conditions as retropharyngeal abscess, foreign body, tumor, asthma, angioneurotic edema, tuberculosis and syphilis.

5. Lynah, H. L.: Influenzal Croup, Am. J. Dis. Child. **18**:238 (Oct.) 1919.

Of 62 patients with nondiphtheritic conditions sent to the hospital, 26, or 42 per cent, had acute simple laryngitis and 24, or 37 per cent, had acute exudative laryngotracheitis, while 13 were classified as miscellaneous. The age distribution of these patients corresponds to that of the diphtheritic series, the greatest number being in the 3 to 5 year group, while the smallest number were under 1 year and over 10 years of age. There is a significantly larger number of males than females in this group.

The mortality for the 62 patients was 40 per cent. This is significantly greater than in the diphtheritic series. There were no deaths in the group with simple laryngitis, but 17 of the 23 with exudative laryngotracheitis died (74 per cent). By age, the percentage mortality was highest among the infants (50 per cent) and in the children between 3 and 5 years of age (57 per cent). It was lowest in those over 10 years of age (15 per cent). There was no difference in percentage mortality by sex. These mortality rates are, of course, not reliable for any statistical comparisons, because of the small number of cases in each unit and because of the unrelated variable factors within each group. A greater number of similar cases must be accumulated before reliable comparisons can be made.

In discussing the treatment of nondiphtheritic laryngitis, therefore, we can only give our personal impressions from the types of cases we have seen, without being able to rule out chance variations in such factors as age, sex, type of case, season and virulence of organism.

Treatment.—In acute simple laryngitis, in which heredity and predisposition may be factors, and in the less common but more serious exudative laryngotracheitis, vigorous attempts at medical treatment should always be tried if time permits. The striking improvement in ordinary "croup" after the administration of ipecac and steam is well known. Ice-packs and oxygen in the more serious of these cases will sometimes tide the patient over borderline obstructive dyspnea. If a skilled surgeon is in constant attendance, ready to intervene, such a procedure may be justified. It must be remembered, however, that asphyxia, once it sets in, is of extremely short duration.

In acute simple laryngitis, the need for mechanical relief is rare, but when edema is a prominent feature, intubation may be justified. Four of our 26 patients with edema received intubation, with no deaths.

In the more serious form, laryngotracheitis, in which fibrinopurulent exudate and blood-tinged tenacious plugs are present in the edematous subglottic areas, the mortality is apt to be high regardless of the method of treatment. Most authors favor tracheotomy for this type, although suction and intubation have recently been advocated by Tolle [4] and Baum.[6]

6. Baum, H. L.: Ann. Otol., Rhin. & Laryng. **36**:472, 1927.

As regards etiology, Streptococcus haemolyticus and Streptococcus viridans are the predominant causative organisms. Less frequently the pneumococcus, staphylococcus and influenza bacillus may be prevalent.[7]

Considering the factors of subglottic edema, repeated accumulations of tenacious exudate low in the trachea and the local and systemic dangers from repeated instrumentation in the presence of a virulent pyogenic infection, low tracheotomy would seem to be the ideal form of treatment. If dyspnea is marked, a tracheal catheter may be inserted until tracheotomy is completed, thereby affording better opportunity for proper technic.

Gittins,[8] in 1932, reported 24 similar cases, with a mortality of 61.5 per cent in the 13 patients who were tracheotomized and 18 per cent in the 11 in whom suction and intubation were used. He is, therefore, of the opinion that intubation and suction are superior to tracheotomy. Tolle [4] reported 22 such cases, with a mortality of 50 per cent. None of her patients was treated by tracheotomy alone. Nine were treated exclusively by suction and intubation, with a mortality of 33 per cent. On this basis, she advocates the use of intubation and suction. Before accurate conclusions can be drawn in regard to this question, a larger series, with ample consideration of age, type and severity, must be investigated.

From the available data of Baum,[9] Tolle, Gittins, Peeler,[10] Cultra [11] and ourselves, there were 35 cases of exudative laryngotracheitis in which tracheotomy only was used. Of these patients, 19, or 54 per cent, died. In 46 reported cases in which suction and intubation were used, 21 patients, or 46 per cent, died. This difference would probably not be significant if comparison could be made with a control series. It is suggested, therefore, that the evidence in support of suction and intubation as a preferred method of treatment is not yet conclusive.

SUMMARY AND CONCLUSIONS

The high institutional mortality from obstructive laryngitis in the United States deserves more careful consideration on the part of those entrusted with the health of children. The aggregate fatality among

7. Bradford, W. L., and Leahy, A. D.: Acute Obstructive Laryngitis; Clinical and Bacteriologic Study in Sixty-Six Cases, Am. J. Dis. Child. **40**:298 (Aug.) 1930.

8. Gittins, T. R.: Ann. Otol., Rhin. & Laryng. **41**:422 (June) 1932.

9. Baum, H. L.: Acute Laryngotracheobronchitis, J. A. M. A. **91**:1097 (Oct. 13) 1928.

10. Hart, V. K., and Peeler, C. V.: Primary Tracheobronchial Diphtheria, Arch. Laryng. **13**:714 (May) 1931.

11. Cultra, G. M., and Streit, A. J.: Texas State J. Med. **26**:364, 1930.

772 reported cases of diphtheritic laryngitis was 24 per cent, and among 81 cases of nondiphtheritic exudative laryngotracheitis, 50 per cent.

Although ignorance, lack of prevention of diphtheria, and delay and carelessness of the laity account for much of this mortality, it is our impression from case histories that greater diligence should be exercised in the early precise differentiation of that group of conditions designated as "croup." Delay in the recognition of impending asphyxia should be avoided. Of 352 patients with "croup" admitted to the Minneapolis General Hospital, 35, or 10 per cent, were moribund on admission. Twenty-eight per cent of the cases referred to the hospital with the diagnosis of diphtheria proved to be nondiphtheritic.

The 255 diphtheritic cases in our series in which treatment was given have been classified as minimal, moderately advanced and advanced on the basis of the extent of the membrane. Forty-six per cent (117) were minimal (edema with little or no membrane), and 20 per cent (52) were moderately advanced. Seven per cent (17) were advanced tracheobronchial cases and 27 per cent (69) were cases of advanced pharyngeal diphtheria with laryngeal involvement.

Direct laryngoscopy was of inestimable value in the diagnosis and treatment in our cases. Training in the diagnostic use of the laryngoscope should be incorporated in graduate study in pediatrics.

A statistical analysis of the data in the diphtheritic cases shows:

1. Of the 255 patients with diphtheritic laryngitis who were treated, 49 died (17 per cent).

2. Diphtheritic laryngitis is significantly more common among males (61 per cent) than among females (39 per cent).

3. It occurs most frequently between the ages of 3 and 5 years inclusively. The next most common age groups are those up to 2 years and from 6 to 10 years. The least common is that above 10 years.

4. Sixty-eight per cent of the patients were 5 years of age or under.

5. The youngest patient was 2 weeks old and the oldest was 60 years of age.

6. The largest number of cases (46 per cent) were of the minimal type and the smallest number (7 per cent) were of the tracheobronchial type.

7. By age groups, the percentage mortality is highest below 5 years and above 10 years. It is lowest between the ages of 5 and 10 years.

8. There is no difference in the death rate of males and females.

9. When, because of previous delay in giving antitoxin, surgical intervention becomes necessary, the percentage mortality increases at least twofold.

10. Intubation is the ideal method of surgical treatment in the minimal cases in which laryngeal edema is the principal obstructive factor. In the minimal cases in which intubation was done only 4 per cent of the patients required further intervention and only 15 per cent died. In the moderately advanced cases with a loose membrane, the necessity for further intervention after intubation increased to 36 per cent, with an increase in mortality to 21 per cent. This was probably due in part to asphyxia from a membrane lodged below the tube.

11. When loose membrane obstructs the air passages, laryngeal suction should be applied first. This reduces the necessity for intubation about 75 per cent, and the percentage mortality in similar cases is reduced to about one half (11 per cent compared to 21 per cent).

12. Including all types of laryngeal diphtheria reported, suction as a first means of relief in selected cases reduced the mortality for the entire group about 10 per cent.

13. Intubation was preferably performed by the direct method, through a laryngoscope.

14. When normal breathing was not restored after three trials of extubation, tracheotomy was performed to prevent chronicity. This operation is particularly to be considered in small infants, in whom fundamental differences in the larynx exist.[12]

In nondiphtheritic laryngitis, the age and sex distribution were the same as in diphtheritic laryngitis. The mortality of the entire group (40 per cent) was significantly higher than that of the diphtheritic series.

In acute exudative laryngotracheitis the mortality is especially high, regardless of the method of treatment. From the available data, the evidence in favor of suction and intubation, instead of tracheotomy, as a preferred method of treatment in this disease is not yet conclusive.

ABSTRACT OF DISCUSSION

Dr. W. Ambrose McGee, Richmond, Va.: Dr. Platou's paper brings to one's attention the seriousness of many apparently simple cases of croup, and should make one exert greater diligence in making differential diagnoses of those groups of conditions referred to as croup. One hardly sees severe diphtheria any more in private practice, but one does see the other type of obstructive laryngitis. In the last year I had 4 such cases due to the hemolytic streptococcus, and the mortality was 50 per cent. The division of the diphtheritic cases into groups, according to the degree of severity, is a practical one and illustrates the seriousness of delay in the proper and prompt handling of those cases. One should not wait for cyanosis, as the mortality at that time is about 90 per cent. A reasonable point stressed by Dr. Platou is that no one method of intervention is the best for all cases. When

12. Tucker, G.: The Infant Larynx: Direct Laryngoscopic Observations, J. A. M. A. **99**:23 (Dec. 3) 1932. Connerty, O.: Deutsche med. Wchnschr. **51**: 175, 1925.

dyspnea is due to edema alone, one might think that intubation rather than suction would be preferable, and in those cases in which there is a loose membrane, suction followed, if necessary, by intubation would be indicated. Training in the use of the laryngoscope should be a part of the pediatric internship.

Dr. Kenneth A. Phelps, Minneapolis: Dr. Platou has attempted to establish a definite form of treatment for a definite type of laryngeal disease. He has classified laryngeal diseases and has applied modern statistical methods to his and other groups of cases. His findings bring out clearly that there is no mathematical evaluation which can be placed on skill, judgment or expert experience.

I should like to emphasize the diagnostic value of direct laryngoscopy. Pediatricians should certainly be able to use a direct laryngoscope. It is not a difficult instrument to use, and it does not take much time. Laryngoscopy is not necessarily a procedure for the hospital; the larynx can be examined directly in the home. It certainly is of value in clinching the diagnosis.

Following Dr. Platou's classification one will know that minimal diphtheria, that is, that without a membrane, requires intubation; moderate diphtheria requires suction and only 25 per cent of cases intubation, and the advanced type, with considerable membrane, requires tracheotomy.

The last point that I shall emphasize is his recommendation of tracheotomy for laryngeal obstruction due to streptococcic or acute infectious laryngitis. In my experience, such patients do not do well following suction or intubation. Tracheotomy should always be done, as both ventilation and drainage can be accomplished through the tracheotomy tube. Suction can be applied by means of a catheter, and the dry, gluelike crusts are removed from the trachea much better than by the intubation tube.

Dr. E. S. Platou, Minneapolis: I have nothing to add except to emphasize the tremendous importance of the impending signs of asphyxia. I do not think that the average medical school emphasizes this sufficiently in its teaching. As Dr. Jackson has stated, when an intern is called about a patient in distress, his response is almost stereotyped: "Is the child cyanotic?" Of course it is well known that when cyanosis appears, the chances of life are very poor.

EFFECT OF VARYING SUGAR INTAKE ON NITROGEN, CALCIUM AND PHOSPHORUS RETENTION OF CHILDREN

REBECCA B. HUBBELL, PH.D.
AND
MARTHA KOEHNE, PH.D.
WITH THE ASSISTANCE OF ELISE MORRELL, M.S., AND BO PRYTZ, M.S.
ANN ARBOR, MICH.

In connection with a group study of the factors concerned in dental caries we have had under observation in the social hygiene unit of the University Hospital children whose food intakes have been accurately determined over periods of from six to eighteen months. Because of the long periods on a controlled food intake, it seemed to us that the group was a desirable one to use for experiments in mineral balance, since the usual period of adjustment would not be necessary.

We have determined the retention of nitrogen, calcium and phosphorus during thirty-five seven day periods, using as subjects girls from 7 to 11 years of age, chosen from the group previously described by Koehne and Morrell.[1] The menus were planned so that the intake of essential constituents of the diet was constant from day to day, and the control ration (diet 1) for the study of metabolism was that which was customarily used on one day of a typical week. This was chosen because it was particularly acceptable to the children and because the average food intakes on this day's menus so nearly equaled the general averages for the entire period of time that the girls were under observation. The diet was adequate as measured by accepted standards, with the following distribution of calories: cereals, bread, etc., 15.8 per cent; milk, 31.8 per cent; vegetables and fruit, 17.1 per cent; fats, 18.1 per cent; meat, eggs, cheese, etc., 11.4 per cent; sugar and sweets, 5.6 per cent. The percentage of calories from protein was 14.3, from fat, 43.2 and from carbohydrate, 42.6. Calcium and phosphorus were estimated to average 1.4 and 1.5 Gm. per day, respectively. This diet was modified by the addition of sugar in the form of candy, to make diets 2 and 3. For diet 2 the added sugar represented a 6 per cent increase in calories, while

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This report represents part of a group study of the cause of dental caries which has been conducted in the School of Dentistry of the University of Michigan on a grant from the Children's Fund of Michigan.

1. Koehne, M., and Morrell, E.: Food Requirement of Girls from Six to Thirteen Years of Age, Am. J. Dis. Child. **47**:548 (March) 1934.

for diet 3 the increase was from 16 to 18 per cent. These rations of higher sugar content were designed to study the influence of sugar on dental caries; and the experiments in balance described in this paper were planned to determine whether an increase in the intake of sugar had any effect on the utilization of calcium and phosphorus. By a comparison with the records of the food intake for several weeks before the periods for studying mineral balance it was evident that the intake of the essential constituents of the diet was not significantly altered. The addition of sugar to the diet resulted only in a higher caloric intake.

Samples of all the foods were analyzed. Nitrogen was determined in the fresh food. For the determinations of calcium and phosphorus the food was dried and then ashed in an electric muffle furnace at a temperature just below dull red heat. The analyses were made of the ash. Feces for the period were marked off in the usual way by carmine, and nitrogen was determined each day in the fresh material. The remainder was dried and ashed, the calcium and phosphorus being determined in the ash. Aliquot portions of twenty-four hour specimens of urine were combined and preserved with toluene. Nitrogen determinations were made twice during the week of study of metabolism. Calcium and phosphorus were determined at the end of the period in the combined aliquot portions. These determinations were also checked by analysis of the ashed urine.

The methods of analysis were the usual standard ones: Kjeldahl's for nitrogen, McCrudden's [2] for calcium, McCandless and Burton's [3] for phosphorus in the food and feces and Epperson's [4] for urinary phosphorus.

Data for all determinations are summarized in table 1 while in table 2 are presented the averages obtained in all experiments for each type of diet used, and in addition, the average distribution of nitrogen, calcium and phosphorus in the urine and feces.

With the standard diet there were the usual variations among the subjects and for the same subject at different times. This was particularly true of nitrogen, owing to a greater variation in intake than was the case with calcium and phosphorus. When extra calories were furnished in the form of sugar there seemed to be a slight effect on the

2. McCrudden, F. H.: The Quantitative Separation of Calcium and Magnesium in the Presence of Phosphates and Small Amounts of Iron Devised Especially for the Analysis of Foods, Urine and Feces, J. Biol. Chem. **7**:83, 1909-1910.

3. McCandless, J. M., and Burton, J. Q.: Sources of Error in the Determination of Phosphoric Acid by the Molybdate-Magnesia Method, J. Indust. Eng. Chem. **16**:1267, 1924.

4. Epperson, A. W.: The Pyrophosphate Method for the Determination of Magnesium and Phosphoric Anhydride, J. Am. Chem. Soc. **50**:321, 1928.

retention of the elements studied. With diet 2, in which the calories derived from cane sugar were increased only 3 per cent, data are available on two children (11 and 21) for five periods. In each case there appeared to be a decided decrease in nitrogen retention. This is proba-

TABLE 1.—*Nitrogen, Calcium and Phosphorus Retention in Children from Seven to Eleven Years of Age* *

Subject	Age Yr.	Age Mo.	Weight, Kg.	Diet†	Preliminary Period on Diet, Days	Nitrogen Intake, Gm.	Nitrogen Excretion, Gm.	Nitrogen Retention, Gm.	Calcium Intake, Gm.	Calcium Excretion, Gm.	Calcium Retention, Gm.	Phosphorus Intake, Gm.	Phosphorus Excretion, Gm.	Phosphorus Retention, Gm.
2	7	0	20.5	1	35	0.405	0.378	+0.027	0.064	0.067	—0.003	0.061	0.054	+0.007
	7	4	21.4	1	142	0.413	0.383	+0.030	0.059	0.051	+0.008	0.060	0.055	+0.005
	7	8	22.7	1	268	0.409	0.390	+0.019	0.054	0.048	+0.006	0.062	0.052	+0.010
5	7	0	25.4	1	6	0.384	0.379	+0.005	0.058	0.049	+0.009	0.058	0.052	+0.006
	7	4	26.2	1	126	0.415	0.391	+0.024	0.060	0.053	+0.007	0.061	0.052	+0.009
6	7	2	25.4	1	50	0.354	0.356	—0.002	0.055	0.053	+0.002	0.060	0.054	+0.006
	7	7	28.0	3	55	0.393	0.364	+0.029	0.053	0.055	—0.002	0.057	0.049	+0.008
13	9	1	26.6	1	6	0.339	0.326	+0.013	0.052	0.043	+0.009	0.049	0.048	+0.001
	9	4	26.9	1	70	0.371	0.355	+0.016	0.055	0.055	±0.000	0.054	0.052	+0.002
9	8	2	27.3	1	61	0.370	0.336	+0.034	0.053	0.057	—0.004	0.056	0.046	+0.010
11	8	2	26.6	1	3	0.408	0.337	+0.071	0.055	0.053	+0.002	0.058	0.048	+0.010
	8	6	27.3	2	65	0.382	0.348	+0.034	0.052	0.049	+0.003	0.058	0.045	+0.013
	8	10	28.2	2	191	0.368	0.344	+0.024	0.058	0.056	+0.002	0.057	0.051	+0.006
	9	5	29.3	2	276	0.371	0.355	+0.016	0.052	0.050	+0.002	0.055	0.049	+0.006
7	7	3	28.0	1	34	0.332	0.239	+0.093	0.057	0.044	+0.013	0.045	0.039	+0.006
31	11	3	29.3	1	8	0.396	0.352	+0.044	0.060	0.043	+0.017	0.069	0.048	+0.021
8	7	7	29.7	1	55	0.343	0.328	+0.015	0.050	0.041	+0.009	0.049	0.042	+0.007
	7	10	32.0	3	84	0.366	0.351	+0.015	0.050	0.046	+0.004	0.054	0.044	+0.010
10	8	2	29.3	1	45	0.348	0.320	+0.028	0.051	0.041	+0.010	0.057	0.050	+0.007
	8	5	32.4	3	56	0.370	0.324	+0.046	0.049	0.044	+0.005	0.053	0.036	+0.017
20	10	0	30.2	1	7	0.378	0.328	+0.050	0.051	0.045	+0.006	0.053	0.043	+0.010
	10	4	30.9	1	114	0.387	0.362	+0.025	0.057	0.047	+0.010	0.058	0.046	+0.012
	10	8	31.8	1	240	0.364	0.354	+0.010	0.053	0.050	+0.003	0.055	0.045	+0.010
16	9	8	32.3	1	61	0.313	0.361	—0.048	0.045	0.052	—0.007	0.048	0.052	—0.004
	10	1	32.2	3	50	0.327	0.293	+0.034	0.048	0.051	—0.003	0.049	0.039	+0.010
30	9	11	31.8	1	96	0.350	0.360	—0.010	0.051	0.050	+0.001	0.051	0.048	+0.003
12	8	9	33.3	3	92	0.335	0.302	+0.033	0.043	0.042	+0.001	0.050	0.041	+0.009
	9	1	35.5	3	210	0.309	0.292	+0.017	0.043	0.037	+0.006	0.046	0.039	+0.007
	9	7	34.0	1	24	0.322	0.303	+0.019	0.043	0.037	+0.006	0.048	0.041	+0.007
19	10	5	38.4	1	13	0.330	0.326	+0.004	0.041	0.034	+0.007	0.045	0.034	+0.011
21	10	10	37.7	1	3	0.318	0.245	+0.073	0.043	0.037	+0.006	0.046	0.035	+0.011
	11	2	40.9	2	71	0.289	0.257	+0.032	0.040	0.032	+0.008	0.044	0.035	+0.009
	11	6	43.3	2	197	0.260	0.250	+0.010	0.039	0.025	+0.014	0.039	0.029	+0.010
24	11	1	39.5	1	20	0.328	0.307	+0.021	0.044	0.037	+0.007	0.049	0.044	+0.005
	11	5	41.4	3	106	0.254	0.242	+0.012	0.040	0.037	+0.003	0.038	0.034	+0.004

* Results are expressed in grams per kilogram per day.
† Diet 1 was well balanced and low in sugar; diet 2 was well balanced, with a small amount of sugar added, and diet 3 was well balanced, with a large amount of sugar added.

bly due in part to the large amount of nitrogen retained during the first week of determinations, which, in each case, followed a short preliminary use of the same type of diet. In view of this short preliminary period, we can hardly attribute the decreased nitrogen retention in the succeeding weeks to the effect of sugar, especially since the amount which was added was so small. The calcium retention of subject 11 was not changed during the period when the diet included increased sugar, while that of subject 21 was somewhat increased. In subject 11

phosphorus seemed to be less well retained when sugar was added, but the values are comparable with those of other subjects on the standard diet and probably are not significant.

Six children (6, 8, 10, 12, 16, 24) were subjects of seven studies using diet 3 which furnished from 20 to 25 per cent extra calories from cane sugar. In the case of subject 12, two weeks on the ration with increased sugar preceded the experimental week during which the standard ration was used. During the first of these periods, with a high intake of sugar, retention of both nitrogen and phosphorus was greater than during either of the succeeding periods, while that of calcium was distinctly lower. There was a considerable drop in body weight for subject 12 during the twenty-four days which elapsed between the second week with the high sugar intake and the determination of the balance with the standard diet. It is possible, therefore, that the figures for this third period do not represent accurately the normal utilization of nitrogen, calcium and phosphorus for this child. With the other five children, retention of nitrogen and phosphorus was increased in three cases (6, 10, 16); retention of nitrogen was unchanged, while that of phosphorus was increased in one (8). In but one instance (24) was there a decrease in nitrogen retention. The phosphorus for this subject was unchanged. In four cases (6, 8, 10, 24) there was a decrease in the amount of calcium retained, while for one (16) there was a slight increase. With the subjects who showed a decreased calcium retention, the loss in every case was through the urine and not the feces, indicating that the excess sugar did not cause a digestive disturbance.

TABLE 2.—*Summary of Nitrogen, Calcium and Phosphorus Retention at Different Levels of Sugar Intake* *

Diet	Number of Determinations	Nitrogen				Calcium				Phosphorus			
		Intake	Excretion		Retention	Intake	Excretion		Retention	Intake	Excretion		Retention
			Feces	Urine			Feces	Urine			Feces	Urine	
1	23†	0.362	0.048	0.288	+0.026	0.052	0.044	0.003	+0.005	0.054	0.021	0.026	+0.007
1	2‡	0.363	0.044	0.247	+0.072	0.049	0.044	0.001	+0.004	0.052	0.020	0.022	+0.010
2	5	0.334	0.043	0.279	+0.012	0.048	0.041	0.003	+0.004	0.051	0.020	0.023	+0.008
1	6‡	0.332	0.039	0.281	+0.012	0.048	0.042	0.002	+0.004	0.051	0.019	0.027	+0.005
3	7	0.333	0.045	0.264	+0.024	0.047	0.041	0.004	+0.002	0.049	0.018	0.023	+0.008

* Results are expressed in grams per kilogram per day.
† An average of all determinations using diet 1.
‡ A comparison of the same subjects given diets 1 and 2 or diets 1 and 3.

SUMMARY

1. Nitrogen, calcium and phosphorus balances were determined for seventeen children, from 7 to 11 years of age. The variable factor in the diet was the level of sugar intake.

2. In twenty-three seven day periods, with a diet low in sugar, an average nitrogen retention of 0.026 Gm. per kilogram per day was obtained, while for calcium and phosphorus the corresponding values indicated a retention of 0.005 and 0.007 Gm. per kilogram per day, respectively.

3. In five seven day periods, with the diet modified only by the inclusion of sugar to give a 6 per cent increase in caloric value, there was no change which could be attributed to the added sugar.

4. In seven periods with the diet modified only by the inclusion of sugar to give an increase of from 16 to 18 per cent in caloric value, there was a tendency toward an increased retention of nitrogen and phosphorus. The retention of calcium was somewhat decreased in five of the seven cases.

BLOOD CELLS IN HEALTHY YOUNG INFANTS

I. THE LEUKOCYTIC PICTURE DURING THE FIRST THREE MONTHS WITH SPECIAL REFERENCE TO HOURLY AND DAILY VARIATIONS

ALFRED H. WASHBURN, M.D.
DENVER

A review of the literature reveals how little accurate information exists concerning the possible variations which may be found in the leukocyte counts of a healthy infant. A brief perusal of the most modern textbooks of pediatrics shows an even greater lack of data in regard to such individual variations. The presentation of average figures for babies of a given age is not very helpful. The assumption that "average" means "normal" for a given organism is pernicious. It has been well established for a quarter of a century that the total number of leukocytes in the peripheral blood stream is subject to considerable fluctuation during the course of a few hours. This fact might well lead one to inquire how great a variation from the average may be consistent with health and what degree of fluctuation points to the presence of some disease process. An additional question is whether there are individual peculiarities demonstrable both in the degree of fluctuation and in the zone within which the total white blood cell counts vary or whether one can assume that all babies have approximately the same variations during periods of good health.

The averaging of many counts destroys not only the picture of the possible variations in a given infant but also the understanding of distinctive leukocytic reactions in different babies. If one is to use the white blood cell count as a diagnostic aid in infancy, this knowledge would seem to be of definite value.

This study of the leukocytes in healthy young infants represents an attempt to discover what variations are consistent with health during the first three months of life. This contribution deals with the hourly fluctuations in the total white blood cell count during different days in the same babies. A preliminary report is made in regard to the simultaneous variations in the differential count. Subsequent contributions from this study will deal with: (1) the more complete details of the differential count, together with descriptions of unusual cell types; (2) variations in the erythrocytes and in the amount of hemoglobin; (3) variations in the platelets and coagulation mechanism.

From the Child Research Council and the University of Colorado School of Medicine.

THE LITERATURE

In spite of the inadequacy of the knowledge as to what constitutes the reflection of good health in the infant's blood picture the literature of the past decade contains many articles which describe the leukocytic picture as "altered" by various diseases, by internal or external irritants, or by drugs, vaccines and the like. With tables of averages borrowed from textbooks or derived from a varying number of single counts on hospitalized babies, the authors of many articles have interpreted fluctuations of from 25 to 50 per cent as being significant of disease or abnormality. There are a number of studies, published during the last few years, which should make one skeptical in accepting such interpretations. The new-born period has received intensive study, and the leukocytic picture is so well established that it will not be considered in this paper. (For these data articles by Lippman,[1] Lucas and his associates [2] and Forkner [3] are suggested.)

While there has been a reasonable degree of consistency in the reported leukocyte counts in new-born babies no such uniformity is shown in the so-called "normal" counts for the next six months of life. Without pretending to review the entire literature on this subject, it may be worth while to quote figures from a few selected authors. Karnizki,[4] in 1903, reported a possible variation of from 8,600 to 16,000; Mitchell,[5] in 1915, from 7,960 to 17,150; Rominger,[6] in 1923, from 9,000 to 23,700; Hoffman,[7] in 1925, from 6,200 to 21,000; Schmal,[8] in 1926, from 7,000 to 23,500; Kovacs,[9] in 1930, from 7,750 to 23,500, and

1. Lippman, H. S.: A Morphologic and Quantitative Study of the Blood Corpuscles in the New-Born Period, Am. J. Dis. Child. **27**:473 (May) 1924.

2. Lucas, W. P.; Dearing, B. F.; Hoobler, H. R.; Cox, A.; Jones, M. R., and Smyth, F. S.: Blood Studies in the New-Born, Am. J. Dis. Child. **22**:525 (Dec.) 1921.

3. Forkner, C. E.: Studies on the Living Blood Cells of the Newborn, Bull. Johns Hopkins Hosp. **45**:75 (Aug.) 1929.

4. Karnizki, A. O.: Ueber das Blut gesunder Kinder, Arch. f. Kinderh. **36**:42, 1903.

5. Mitchell, A. G.: Leukocyte Counts During Digestion in Bottle-Fed Infants, Am. J. Dis. Child. **9**:358 (May) 1915.

6. Rominger, E.: Nutritional Disturbances and Blood, Jahrb. f. Kinderh. **103**:1 (Sept.) 1923.

7. Hoffman, A., and Welcker, A.: Shifting to Left in Blood Picture in Diagnosis and Prognosis in Healthy Infants, Arch. f. Kinderh. **76**:1 (April) 1925.

8. Schmal, S.; Schmidt, W., and Serebrijski, J.: Leukocytes in Infants, Premature Infants and Small Children. Normal White Blood Picture in Infants and Premature Infants, Ztschr. f. Kinderh. **41**:102, 1926.

9. Kovacs, E.: Studies of Leukocyte Counts in Nurslings, Jahrb. f. Kinderh. **128**:244 (July) 1930.

Smith,[10] in 1930, from 6,000 to 16,000. The average figures in such standard textbooks as Holt and Howland's,[11] Lucas',[12] Abt's "Pediatrics," [13] or "Clinical Pediatrics," [14] range from 10,000 to 15,000. If the variations noted by the authors just mentioned were charted, the total leukocyte counts in infants apparently free from disease would range from 3,800 [15] to 23,700.[6] Assuming that these figures represent accurate findings on healthy infants it would seem to be high time to revise one's ideas, as well as one's books, concerning the significance of counts below 8,000 and over 15,000. Furthermore, it might be expected that the intensive study of a small group of babies would give more information than an equal number of single counts on many infants.

This method of approach has already been utilized in a number of studies. In 1915, Mitchell,[5] studying the effect of digestion on the leukocytic picture, published interesting figures showing the fluctuations in the white blood cell counts every fifteen minutes for the three to four hours between feedings. Owing to the fact that he averaged most of his counts by a series or by intervals of time after feeding, it is not possible to compare his published results with those of the present study. In spite of this minimizing of the individual characteristics, the variations which we found were similar to those reported later. Mitchell [5] quoted Wernstedt to the effect that such serial counts, when charted, have much the same appearance as irregular fever curves.

In 1926, Sabin and her co-workers [16] studied the hourly leukocyte fluctuations in young adults, and in 1927, Shaw,[17] in England, carried out a similar study. Both groups of workers arrived independently at the conclusion that there are hourly "rhythms" or "tides" which can be demonstrated in the total number of white blood cells in circulation. The proof of this contention would require the following of a group of

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10. Smith, C. H.: Differential White Count in Infancy. Comparison by Supravital and Fixed-Smear Method, Am. J. Dis. Child. **40**:505 (Sept.) 1930.

11. Holt, L. Emmett, and Howland, John: Diseases of Infancy and Childhood, New York, D. Appleton and Company, 1928.

12. Lucas, William Palmer: Modern Practice of Pediatrics, New York, The Macmillan Company, 1927.

13. Lucas, W. P., and Fleischner, E. C.: The Physiology of the Blood, in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 4.

14. Lucas, W. P., and Washburn, A. H.: Diseases of the Blood and Blood-Building Organs. Clinical Pediatrics, New York, D. Appleton and Company, 1928.

15. Fletcher, E. G., and Mitchell, A. G.: Physiologic Variations in the Leukocytes in Infants and in Children, Am. J. Dis. Child. **34**:807 (Nov.) 1927.

16. Sabin, F. R.; Cunningham, R. S.; Doan, C. A., and Kindwall, J. A.: The Normal Rhythm of the White Blood Cells, Bull. Johns Hopkins Hosp. **37**:14 (July) 1925.

17. Shaw, A. F. B.: Diurnal Tides of Leukocytes of Man, J. Path. & Bact. **30**:1 (Jan.) 1927.

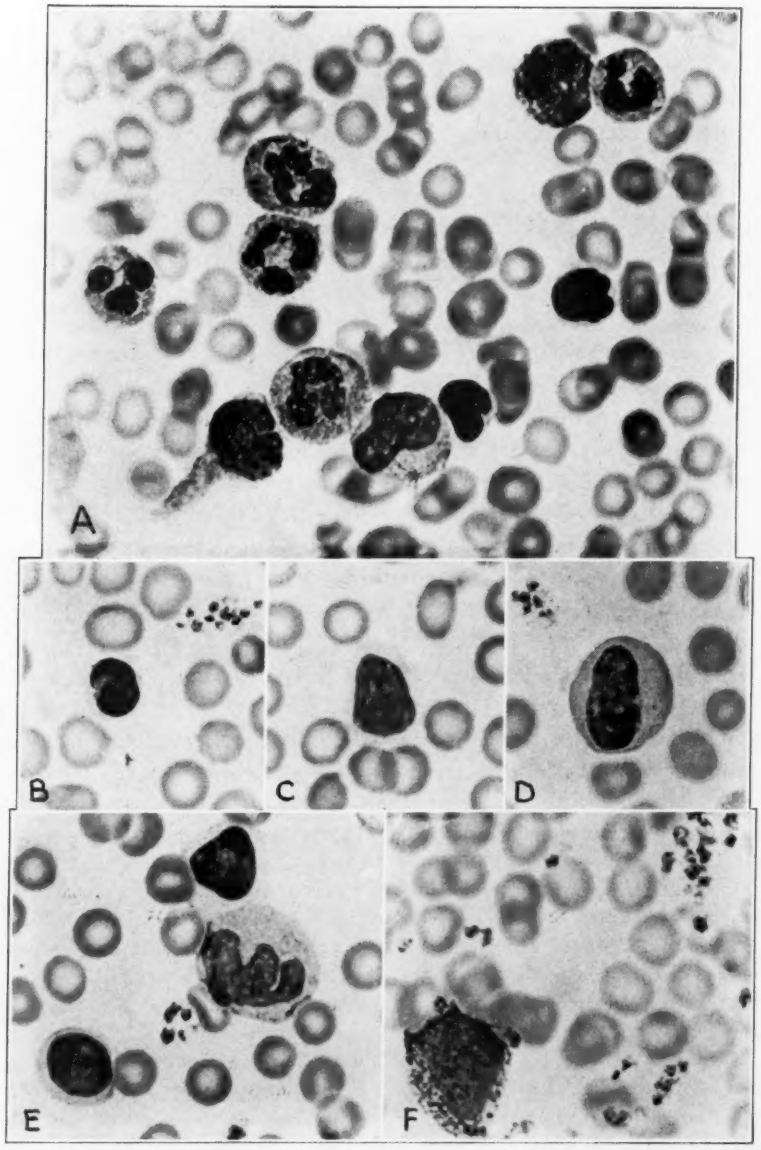

Fig. 1.—Cell types: *A*, neutrophils of varying degrees of maturity; *B*, small lymphocyte; *C*, large lymphocyte; *D*, (lympho ?—) blast; *E*, large mononuclear cell with two typical lymphocytes (intermediate); *F*, unclassified cell with granules resembling those in platelets.

persons through several different twenty-four hour periods, demonstrating that the high and low points of the leukocyte curves tended to recur at approximately the same hours each day. Without entering into the controversy in regard to the presence of such rhythmic fluctuations in the adult I feel safe in stating that there is no good evidence of such regular fluctuations in the case of small infants. In 1927, Fletcher and Mitchell [15] reported hourly leukocyte counts on four small infants for

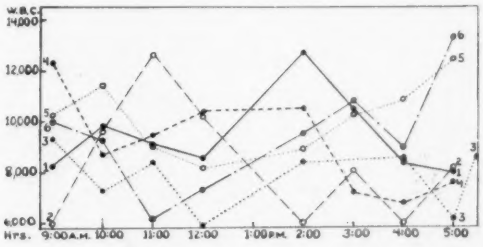

Fig. *2* (case 9, boy).—Curves *1* to *5* represent the total white blood cell counts done at 1, *2*, 3, 4, 5 and *12* weeks of age.

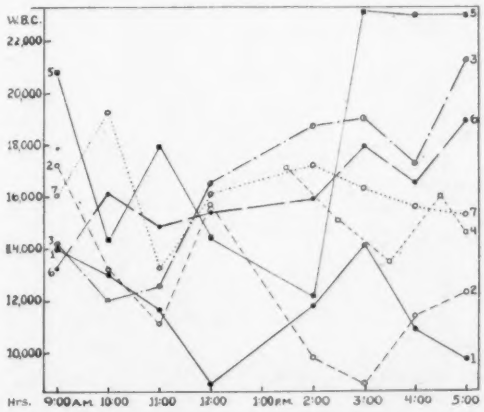

Fig. *3* (case 10, girl).—Curves *1* to *7* represent the total white blood cell counts done at *2*, 4, 5, *7*, 12, 13 and 14 weeks of age. Both the degree of variability by the hour and the individual zones within which the leukocyte counts fluctuate should be compared in these two cases.

one period of twenty-four hours. The curves constructed from these counts are similar to some presented in this paper. Their conclusions that "the times of day and night are factors to be considered in the evaluation of leukocyte counts in infants and in children" and that "blood counts should be made at the same hours on successive days" do not seem warranted from the data given. In their published curves,

high peaks occurred at such widely varied hours as 1, 4, 6 and 10 a. m., 2 and 3 p. m. and midnight, while low points were found at 6, 7, 8 and 9 a. m. and 1, 4, 9 and 11 p. m. That repeated series on the same infants would have shown even greater irregularities is suggested by the studies of Kovacs [9] on eight babies between the ages of 1 and 10 months. Counts were done at 6 and 10 a. m. and at 2 and 6 p. m. on each baby for two successive days. The difference between the lowest and highest

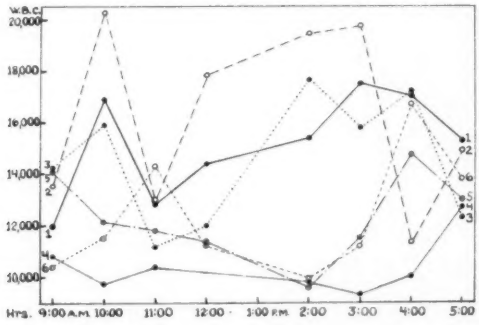

Fig. 4 (case 11, girl).—Curves *1* to *6* represent the total white cell counts done at 2, 4, 6, 8, 10 and 12 weeks of age.

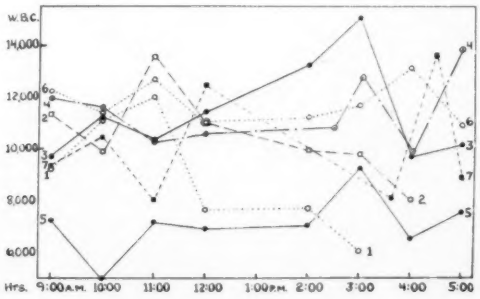

Fig. 5 (case 12, girl).—Curves *1* to *8* represent the total white cell counts done at 1½, 3½, 5½, 7½, 10½, 12½, 14½ and 16½ weeks of age. The contrast between the zone covered by the leukocyte counts and the percentage of increase from the lowest to the highest counts recorded is notable.

counts on any given baby ranged from 30 to 125 per cent, and in one instance there was an increase of 125 per cent from the count made at 2 p. m. on one day to the count made at 2 p. m. on the following day. A study of all the figures given by Kovacs also gives some support to the hypothesis previously mentioned, that there may be individual characteristics apparent in the maintenance of a given leukocyte level or zone within which the hourly fluctuations occur. In this same connection

the figures given by Martin [18] for young women during rest and after exercise or after the injection of epinephrine are also of interest.

There is a considerable volume of literature concerning the differential white blood cell count in infancy and during childhood. Many authors report their figures in terms of percentages, while some fail to note the total leukocyte count at the time the smear was made. With fluctuations of from 15 to 75 per cent in the total number of white cells within two to three hours it must be apparent that the presentation

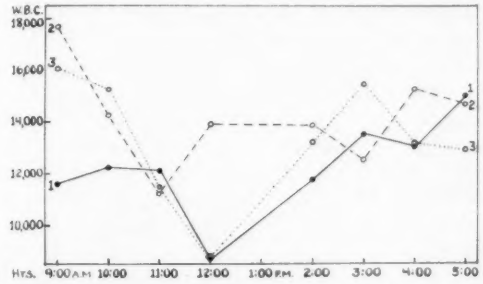

Fig. 6 (case 13, boy).—Curves *1*, *2* and *3* represent the total white blood cell counts done at 1, 3 and 5 weeks of age.

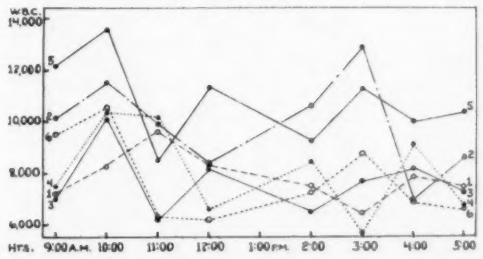

Fig. 7 (case 14, boy).—Curves *1* to *6* represent the total white blood cell counts done at 1½ 3⅓, 4½, 5½, 6½ and 8½ weeks of age. Curves *1*, *2* and *4* should be compared with curves *1*, *2* and *3* in figure 6.

of percentages of the different types of cells will not give a fair portrayal of the leukocytic picture. Thus with a rise from 10,000 to 15,000 white cells the percentage of neutrophils may drop from 20 to 15, but that will represent an actual increase in their total number from 2,000 to 2,250 per cubic millimeter. The figures given by Fletcher and Mitchell [15] show a variation in the number of neutrophils from 500 to 8,500 per cubic millimeter and of lymphocytes from 2,000 to 10,500.

18. Martin, H. E.: Physiologic Leukocytosis; Variations in the Leukocyte Count During Rest and After Exercise and After Hypodermic Injection of Adrenalin, J. Physiol. **75**:113 (June) 1932.

With the routine technic, with Wright's stain on fixed smears, these figures are probably as reliable as any in the literature. Fletcher and Mitchell, as well as others, have noted the tendency for the curve of the lymphocytes to parallel the curve of the total white blood cell counts more closely than that of the neutrophils.

The importance of vital staining for obtaining accurate differential counts has been stressed by several workers, notably Sabin,[16] Forkner,[3] Smith,[10] Benjamin and Ward [19] and Rogers.[20] In a subsequent report

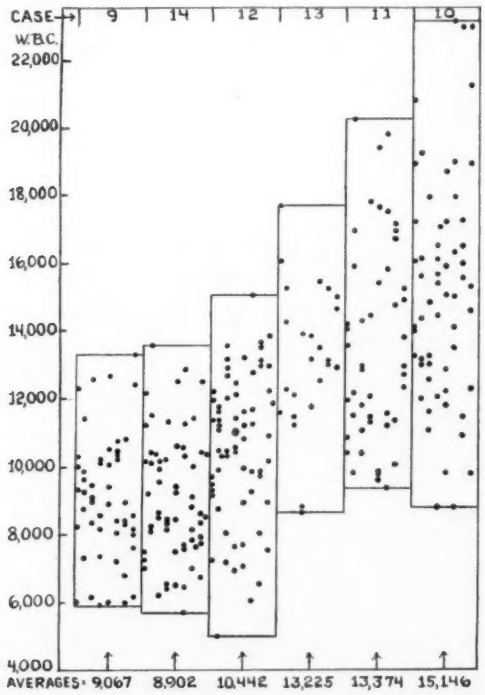

Fig. 8.—The total leukocyte counts done from 1 to 16 weeks of age for cases 9, 14, 12, 13, 11 and 10. The similarity in range between cases 9 and 14 and the marked contrast between these and cases 10 and 11 are notable. The lack of any significance in the average count in relation to the possible individual variations is also demonstrated.

this question will receive more careful consideration. In the present study, dealing largely with total leukocyte counts, a reasonable degree of accuracy will be assumed for the differential counts, which are made on fixed smears stained with Wright's stain as a routine.

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19. Benjamin, B., and Ward, S. M.: Leukocytic Response to Measles, Am. J. Dis. Child. **44**:921 (Nov.) 1932.

20. Rogers, P. M.: A Study of the Blood Monocytes in Children with Tuberculosis, New England J. Med. **198**:740 (May 24) 1928.

RATIONALE AND METHODS OF STUDY

This study was started early in 1931, its purpose being to follow the entire blood cell picture at weekly intervals during the first six months of life. Counts of the erythrocytes, leukocytes and platelets were to be done once a week on each of a group of babies. At the same time the hemoglobin content, volume of red cells and differential leukocyte counts were to be followed by a variety of technics. In spite of the fact that the counts were all taken at approximately the same hour in the morning each week, the white blood cell counts showed extreme variations not only in different babies but in the same baby from week to week. This finding led to checking for several months all possible errors in technic, including those dependent on the apparatus and those concerned with the person counting. Most of these factors had been studied thoroughly by me some years ago, but were rechecked under the conditions of the present study. Since I had no assistants and performed every task myself, this checking of errors was simple and complete. It became apparent that the same healthy baby, day by day or week by week, would show a considerable degree of variation in the number of white blood cells between the hours of 8:30 and 9:00 a. m. In contrast the red blood cell count and hemoglobin percentage showed little variation from day to day, and only relatively slow and, for the most part, consistent changes from week to week. Therefore subsequent attention was focused on the leukocytes in the effort to obtain an accurate picture of the variations which might be consistent with average health during the hours of the day when babies' blood counts would usually be obtained.

The babies utilized in this study were at the Florence Crittenton Home. They were kept in the nursery where their feedings and entire twenty-four hour regimen could be maintained as nearly constant as possible. The daily routine in the nursery was as follows:

6 a. m., nursery lighted as day nursing shift arrived and babies began to be disturbed; 6:30, feeding; 8:00, orange juice followed by a bath and fresh clothes; 9:30, feeding and cod liver oil; 12:30, 3:30, 6:30 and 10:30 p. m. and 2:30 a. m., feedings.

The majority of the babies in the nursery were on a diet of a mixture of ordinary whole milk, water and dextrimaltose made up with 3 parts of milk, 1 part of water and sufficient dextrimaltose to furnish from 45 to 50 calories per pound (453.6 Gm.) of weight per day. Careful records were kept as a check on the health and actions of each baby. No abnormal or sick babies were included in the series studied, and every effort was made to exclude all but averagely healthy babies on an average pediatric regimen. For each of the six infants who form the material for this report hourly leukocyte counts were done between 9 a. m. and 5:30 p. m. at intervals varying between three days and four weeks. The majority of the counts were taken at 9, 10, 11 a. m. and noon, and at 2, 3, 4 and 5 p. m., giving a total of eight counts with a rest period of two hours from noon to 2 p. m. The most frequent interval of time between series was one to two weeks. Smears were made as soon after filling the counting pipet as possible, but were not stained or counted until the following day in order to avoid haste, with its resulting inaccuracies in the classification of cells.

Red blood cell counts and hemoglobin determinations (according to the Sahli technic) were performed once each day. Scattered platelet counts were done in the counting chamber as a check on the number of thrombocytes in the stained smear. Since no significant changes were demonstrable in these three determinations during the day, their repetition during any period of twenty-four hours was discontinued after the first few cases had been studied.

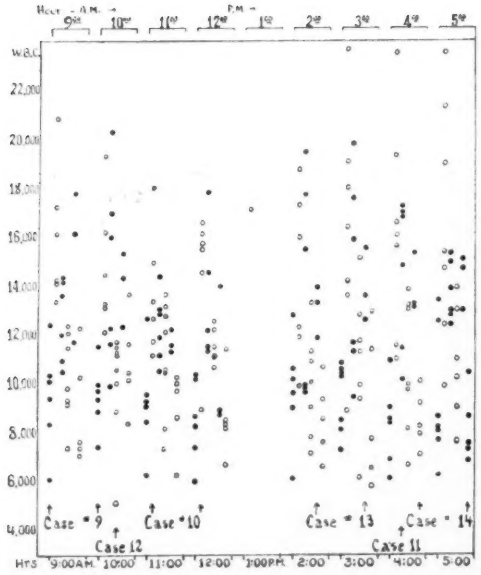

Fig. 9.—The total leukocyte (270 hourly) counts for six infants, ranging in age from 6 days to 16 weeks. When all the counts for cases 9 to 14 are scattered by hours of the day, no tendency to any orderly rhythm of fluctuation is demonstrable.

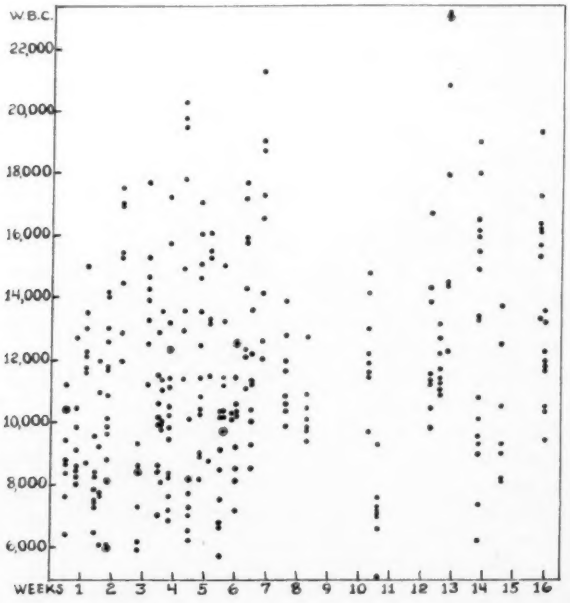

Fig. 10.—The total leukocyte counts by age for six infants ranging in age from 3 days to 16 weeks. When all counts are scattered according to age, no tendencies can be noted that depend on variation in age between 1 and 16 weeks.

TECHNIC AND LIMITS OF ERROR

In every case the blood was drawn from the infant's heel. After checking and finding no gross error involved, it was taken from alternate heels each hour. After obtaining a free flow of blood, the first two or three drops having been sponged off, the pipet for making the white cell count was filled in the usual manner (blood to 0.5 c.mm. and acetic acid to 11 c.mm. to give a dilution of 1 : 20). Cover-slip smears were made as soon as possible after filling the pipet. The pipet was shaken vigorously for at least four minutes by the clock, this interval having been determined as the time required to produce an even suspension of the white cells. A few drops were allowed to run out before filling the first side of the counting chamber and again before filling the opposite side. The leukocytes in the four large corner squares (1 sq. mm. each) on each side of the chamber were counted (a total area of 8 sq. mm.). When the total number of cells in four squares was 200 or less, no counts were accepted if the difference between the total of the four squares on the two sides of the counting chamber

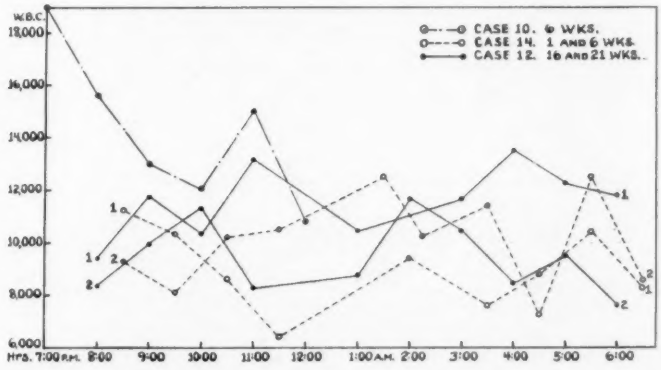

Fig. 11.—The total leukocyte counts taken hourly during the night in cases 10, 12 and 14. The degrees of fluctuation and the zones within which they occur should be compared with the data in figures 3, 5 and 7.

exceeded 10 per cent of the smaller of these two totals. With counts over 200 cells (i. e., a total white blood cell count of over 10,000 per cubic millimeter) the maximum variation allowed between the counts on the two sides of the counting chamber was 20 cells. In the majority of the counts a four minute shaking of the pipet resulted in a distribution of cells so that the difference between the 2 counts did not exceed 10 cells. Occasionally, when the difference was slightly greater than the accepted limit of error the pipet was reshaken and the count repeated. On a few occasions there were gross errors necessitating the discarding of the whole count. A mechanical counter was used for all counts, personal bias being further reduced by obtaining the total count on each side before checking individual squares against each other for evenness of distribution.

Possible errors dependent on minor variations in the rate of blood flow, moderate changes of the temperature in the feet and congestion from pressure about the leg were checked many times but found to be negligible. The opening of a prick made two hours previously was found to give an accurate count, provided a free flow of blood was obtained. In fact, even with gentle pressure to start the flow the count consistently checked with one made simultaneously from a fresh prick, at least within the counter's accepted limits of error. Hence it was

seldom necessary to make a fresh prick every hour. The pipet which was employed for over 90 per cent of the counts has been used by me for the past fifteen years, and has been checked for accuracy. The same was true of the counting chamber. Since I did all my own technical work, "personal equation" errors may be assumed to be fairly constant, if present. In general, it may be said that fifteen years' use of the same apparatus by the same person probably reduced possible errors from faulty technic to a minimum.

The cover-slips used in making the smears were cleaned, polished and kept free from grease or dust. Smears were made in the routine manner with two cover-slips, thin smears being preferred because of the minimum distortion of individual cells, together with the maximum evenness of distribution which results from this technic (fig. 1). The smears were stained with Wright's stain. The two cover-slips were examined to make sure of approximate evenness of distribution. On the better preparation 200 white cells were counted, care being taken to move to a different part of the smear for the second hundred. It was found

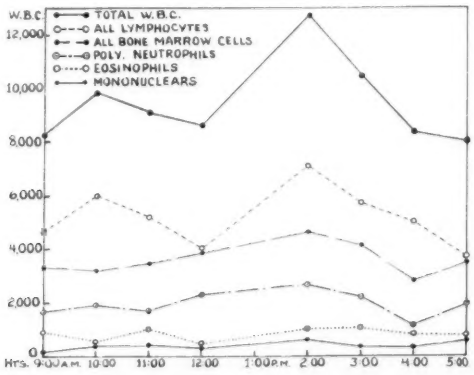

Fig. 12 (case 9).—Differential counts for an infant aged 6 days. The parallelism between the curves for the total leukocyte counts and the lymphocytes is in marked contrast to the absence of any such relationship in the other curves.

that the error was not significantly greater for 200 than for 400 cells, providing the two different hundreds checked fairly closely (according to Barnett [21] the difference in chance error between counts of 200 and 400 cells amounts to ± 3.5 per cent). Gross discrepancies in the 200 led to a discarding of the differential count, while minor differences were checked by counting another 100 to 200 cells. The oil immersion lens was used for classifying all leukocytes.

Every effort was made in method and technic to keep to procedures which are applicable to routine practice, avoiding equipment or situations which could not be duplicated in the average practice of pediatrics.

RESULTS OF THE STUDY

The results of such a study of leukocyte counts can be presented more easily by charts than by words. Figures 2 to 7 show curves constructed from the hourly leukocyte counts on different days on the six

21. Barnett, C. W.: The Unavoidable Error in the Differential Count of the Leukocytes of the Blood, J. Clin. Investigation **12**:77 (Jan.) 1933.

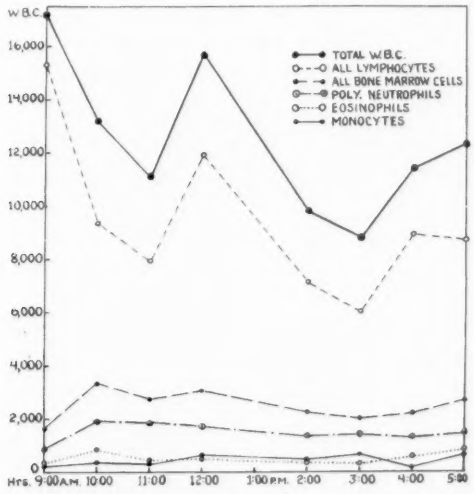

Fig. 13 (case 10).—Differential counts for an infant aged 4 weeks. The striking parallelism between the number of white blood cells and the number of lymphocytes is notable.

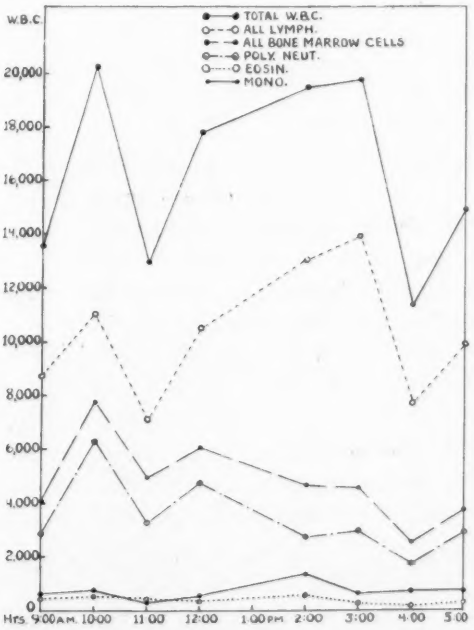

Fig. 14 (case 11).—Differential count for an infant aged 1 month. The difference in the correlation of curves during the morning and afternoon hours is striking.

babies in this report. Since the feeding schedules and daily regimens for all babies were practically identical, the times of these procedures have not been marked. The striking variations on different days on the same baby (figs. 3 and 4) as well as on different babies at the same age (figs. 2 and 3) demonstrate quite clearly the lack of correlation between the leukocyte count and such factors as feedings (Mitchell,[5] Gyllenswärd [22] and Garrey [23]), the daily bath or other routine events of the day. Careful records of the baby's day in each case failed to show any consistent reaction to such additional factors as crying or increased activity (Martin,[18] Garrey [24] and Gonnelle [25]), sleep, sun-baths and moderate changes of temperature in the nursery. One day's record of case 10 (fig. 3, curve 5) may be given to illustrate this lack of correlation. The schedule, reactions and leukocyte counts were as follows:

	Activity	White Blood Cell Count	
8- 9 a. m.	Orange juice, bath and dressing	9 a. m.	20,800
9-10 a. m.	Quiet 9 to 9:30; fed 9:30 to 9:50 (awake and active)	10 a. m.	14,350
10-11 a. m.	Awake most of hour, but quiet	11 a. m.	17,950
11-12 noon	Slept second half of hour	12 noon	14,450
12- 1 p. m.	Fed 12:30 to 1		
1- 2 p. m.	Slept quietly entire hour	2 p. m.	12,200
2- 3 p. m.	Slept quietly entire hour	3 p. m.	23,150
3- 4 p. m.	Slept 3 to 3:30; fed 3:30-4	4 p. m.	23,000
4- 5 p. m.	Awake most of hour; fairly quiet	5 p. m.	23,000

Sometimes an hour of peaceful sleep might be followed by a sharp rise, while at other times it preceded an equally sharp drop, in the leukocyte count. The same thing held true for hours during which there was unusual activity like feedings and baths.

The absence of any rhythms (Fletcher [15] and Sabin [16]) or tides [17] in the number of white cells appearing in the blood stream will be equally apparent from the briefest study of these charts. The danger of drawing conclusions from any one series of hourly counts is well illustrated in all the charts except that for case 13 (fig. 6); this infant was removed from the home after three series had been obtained. In this case two of the curves have considerable similarity, but further study would probably have revealed the same lack of consistency shown

22. Gyllenswärd, C.: The White Blood Picture During Starvation and After Food, Acta pædiat. (supp. 1) **10**:1 (Oct. 31) 1930.

23. Garrey, W. E., and Butler, V.: Digestive Leukocytosis Question, Am. J. Physiol. **100**:351 (April) 1932.

24. Garrey, W. E.: Basal Leukocyte Count and Physiologic Leukocytosis, Proc. Staff Meet., Mayo Clin. **4**:157 (May 15) 1929.

25. Gonnelle, H., and Warter, J.: De l'influence d'un effort musculaire intense (course) sur l'image blanche du sang périphérique, Arch. d. mal. du cœur **23**:757 (Dec.) 1930.

by the other five cases. Aside from the apparent chaos of these leukocyte curves, as shown particularly by comparing figures 2 and 3 or figures 4 and 5, certain other characteristics are worth noting.

Comparison of the cases in the zone within which leukocyte fluctuations occurred shows striking individual characteristics. Thus, case 9 (fig. 2) shows all the counts between 5,900 and 13,300, while case 10 (fig. 3) shows a variation from 8,800 to 23,150. A study of all six charts shows that cases 9 and 14 are the only ones which cover even approximately the same zone. The actual ranges covered by the counts of the different babies vary from 7,400 for case 9 to 14,350 for case 10. When they are treated in terms of percentage increase from the lowest to the highest counts obtained in each case, the difference is less striking but of

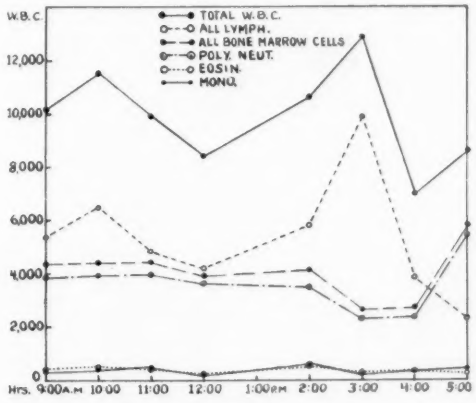

Fig. 15 (case 14).—Differential counts for an infant aged 3 weeks. This curve shows one of the few times when the curve of the neutrophils crossed above that of the lymphocytes.

interest. Case 11 (fig. 4) shows the smallest increase, with 116 per cent, while case 12 (fig. 5) shows the maximum increase, 200 per cent. The different zones occupied by the leukocyte counts in all six cases are shown graphically in figure 8. The averages of all counts on each baby are given at the foot of each column representing the individual ranges.

As a further check on the hourly rhythms the 272 white blood cell counts done between the hours of 9 a. m. and 5 p. m. have been combined in figure 9. Surely no tendency to rhythmic fluctuations is demonstrable in this scattering of the counts on all six babies for the hours of the day. The possibility that age played some part during the first sixteen weeks of life was checked by scattering the total of 291 counts by age in figure 10. The larger number of leukocyte counts performed during the first seven weeks of life makes it unwise to draw

any too definite conclusions. If there is any trend demonstrable, it consists in a slight tendency for the average level to rise during the first six or seven weeks.

The possible variations in the leukocyte counts during the hours of the night have not been investigated thoroughly. Case 10 was followed from 7 p. m. to 12 midnight on one occasion. Cases 12 and 14 were followed through two nights, from 8 p. m. to 6 a. m. The curves constructed from these counts (fig. 11) furnish at least suggestive evidence that the leukocyte fluctuations during the hours of the night do not differ in any essential way from those observed during the day. Further investigation may necessitate some modification of this statement.

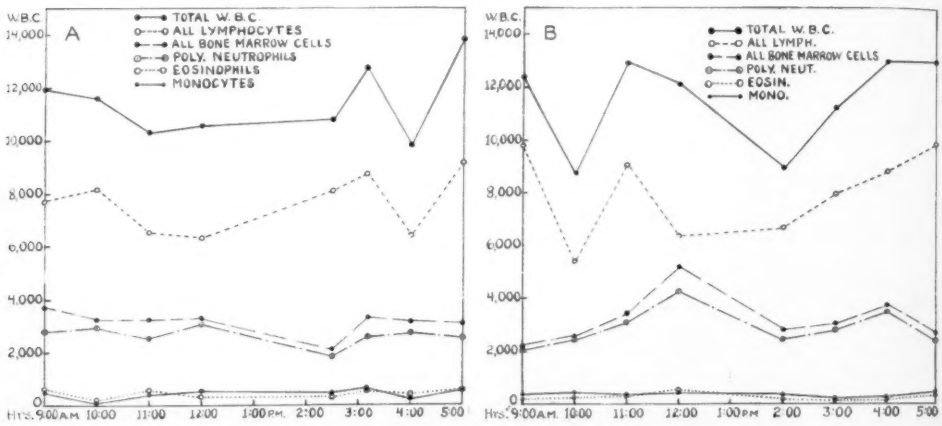

Fig. 16 (case 12).—Differential counts for an infant at the ages of (*A*) 2 months and (*B*) 4 months. These curves illustrate the persistent tendency for the curve of the lymphocytes to follow that of the total white blood cell counts, even when the fluctuations are relatively slight.

Figures 12 to 17 show graphically the variations in the different types of white blood cells in cases 9, 10, 11, 12 and 14. For the sake of clarity in terminology photomicrographs of the various cell types are shown in the figure. The cells having their origin in the bone marrow were separated into mature and immature polymorphonuclears, myelocytes, eosinophils and basophils. Those of lymphoid origin were grouped as large and small lymphocytes, "large" being applied roughly to all those which were approximately two times or more the size of a red blood cell. The monocytes and other large mononuclear forms, exclusive of the lymphocytes, were placed under the one heading of monocytes. No attempt was made to differentiate between lymphoblasts and myeloblasts, such primitive cells being designated simply as "blasts." Usually from 0.5 to 3 per cent of the cells remained "unclassified."

These were all large mononuclear forms which did not fall into any of the routine classifications and were probably immature cells of various kinds. On several smears occasional cells were seen which resembled megakaryocytes (fig. 1 *F*).

The curves for the various types of white cells fail to show any close parallelism except in the case of those for the lymphocytes and for the total leukocyte counts. These two curves are so consistently similar as to suggest that at this age sudden changes in the white cell count are apt to be due to fluctuations in the lymphocytes rather than to changes in the total number of any of the other cell types. Occasionally the curve for the polymorphonuclear neutrophils shows a tendency to follow that of the total white blood cells counts (fig. 14, from 9 to 11 a. m.; fig. 17, from 4 to 5 p. m.). More frequently the curves for all cells except the lymphocytes fail to show any consistent rela-

Extremes of Variation of All Leukocytes *

Cell Type	Minimum	Maximum
Total leukocyte count	3,600	23,150
Mature polymorphonuclears	590	6,277
Immature polymorphonuclears	0	1,066
All polymorphonuclears	885	7,077
Myelocytes	0	1,060
Eosinophils	0	1,805
Basophils	0	204
All bone marrow cells	1,167	7,795
Large lymphocytes	922	10,836
Small lymphocytes	602	12,435
All lymphocytes	2,322	19,090
Large mononuclears	0	1,400
Lymphoblasts or myeloblasts	0	404
Unclassified cells	0	395

* The minimum and maximum numbers per cubic millimeter for all the different types of leukocytes classified in 200 differential counts on ten babies.

tionship to the total leukocyte curve. In order to avoid confusion the curves for immature neutrophils, myelocytes, blasts and unclassified forms have been omitted. In a subsequent report the entire differential picture will be presented in detail. It is possible that some further tendencies may be noted when the whole series has been analyzed more thoroughly. The table shows the minimum and maximum figures observed for all types of white blood cells in 200 differential counts on ten babies whose blood has been followed at hourly intervals.

In reviewing the literature it has been noted that total leukocyte counts from 3,800 to 23,700 have been reported for healthy babies. It is of particular interest to note that the variations given in the table are almost identical with these figures, even though mine were obtained from a study of only ten babies while those from the literature represent the result of counts on hundreds of babies in many parts of the world. This makes it seem probable that the variations given for the differential counts also furnish a reasonably accurate picture of their

possible limits, even though the series is so small. The likelihood that Denver's altitude of 5,000 feet may play some part in producing this blood picture would seem remote in view of the similarity between the leukocyte counts herein reported and those quoted from the literature. Continued intensive study of individual babies may lead to a clearer concept of both the possible variations in the leukocytic picture and their underlying causes.

SUMMARY AND CONCLUSIONS

The hourly fluctuations in the leukocyte counts of six healthy infants have been studied on different days during the age period of 1 to 16 weeks.

Most of the 291 counts have been done between the hours of 9 a. m. and 5 p. m., with a few during the remaining sixteen hours.

The curves showing the leukocyte fluctuations are extremely irregular, showing considerable variability for the same baby during any one day, for the same baby at the same hour on different days and for different babies of the same age and size and at the same hour.

The smallest variation observed for any one baby during the whole period of observation was from 5,900 to 13,300, while the greatest was from 8,800 to 23,150. In terms of percentage increase from the lowest to the highest count, the smallest variation was 116 per cent and the greatest was 200 per cent.

A preliminary report is made concerning the behavior of the different types of white blood cells during the fluctuations in the total count. There is a demonstrable tendency for the curve of the lymphocytes to parallel that of the total leukocyte count.

The following conclusions are suggested, pending the outcome of further study of healthy infants during the first few months of life.

1. Leukocyte counts may vary between 4,000 and 23,000 per cubic millimeter without indicating the presence of any demonstrable disease process.

2. The leukocyte counts in any individual baby tend to fluctuate within a definite zone, but this zone is not the same for every baby of a given size or age.

3. There is no evidence of orderly rhythm in the fluctuations of the leukocyte count during the hours of the day or night. Hence there is no logical reason for expecting counts done at the same hour on different days to be comparable.

4. There is no demonstrable correlation between the variations in the number of white cells and such factors as the intake of food, digestion, sleep, increased activity or minor external disturbances.

5. The cell type which is predominantly responsible for the sharp fluctuations in the total white blood cell count is the lymphocyte.

ATELECTASIS AND BRONCHIECTASIS IN CHILDREN

A STUDY OF FIFTY CASES PRESENTING A TRIANGULAR SHADOW AT THE BASE OF THE LUNG

WILLIAM E. ANSPACH, M.D.

CHICAGO

Clinical and necropsy observations in relatively advanced cases have been the basis for the many theories regarding the development of bronchiectasis. Little has been known about its pathogenesis. No prebronchiectatic conditions have been recognized, and no serial biopsies have been recorded to show the changes from one period to another. Even if advisable, serial biopsies would most likely not reveal a true picture after the first tissue was removed. If, on the other hand, a large series of children with bronchiectasis were found to show a common, almost characteristic appearance in roentgenograms of the chest during the early period of clinical symptoms, a means would be at hand for the recognition of the onset of a bronchiectatic process, possibly before the actual dilatations of the bronchi had occurred. The pathologic changes in the cases that came to necropsy soon after the presence of this sign and subsequent roentgen observations in the patients who lived would reveal much about the development of bronchiectasis. It has been my privilege to make a study of an unusually large series of children presenting a certain triangular shadow at the base of the lung in pulmonary roentgenograms. This shadow has come to be regarded as representing a change in the pulmonary tissue in which, under certain conditions, bronchiectasis develops. In the early roentgenograms of more than fifty children with proved bronchial dilatations, this shadow was found to be present within a few hours or days after the onset of the first symptoms.

From the Children's Memorial Hospital.

Read in part at the Joint Meeting of the Central States Pediatrics Society and the American Academy of Pediatrics, Chicago, Sept. 23, 1933.

Roentgenograms and charts setting forth ideas here presented were exhibited at the meeting of the American Medical Association, Milwaukee, June, 1933; at the American Congress of Radiology, Chicago, September, 1933, and at the International Medical Assembly of the Interstate Postgraduate Medical Association of North America, Cleveland, October, 1933.

Speculation as to possible structures accounting for this triangular shadow on roentgenograms has been recorded by many authors who saw their patients only during a single stage and formed their opinions without postmortem observations or an understanding of the early course of the process. One author,[1] after aspirating only a small amount of serous fluid and 100 cc. of air, thought that the triangular shadow was accounted for by a pneumothorax within a markedly thickened enveloping mediastinal pleura. Because of the fluid-like density of the shadow, others [2] have assumed that they were dealing with pockets of fluid in the mediastinal pleural space or within the mediastinal space itself. Still others [3] failed to differentiate the cases showing the triangular shadow, about to be described, from other cases of "mediastinal pleural effusions." In one of the most recent reports the small clearly defined triangular shadow was attributed to an anomalous lobe.[4] In a textbook [5] a triangular shadow has been shown beneath an aneurysm of the aorta. The assumption of the authors was that it was due to the "central tendon of the diaphragm drawn obliquely along the heart border." Other authors by either description or illustration have pictured the triangular shadow with interpretations of one of the aforementioned conditions. The lack of proof in recorded cases such as these has done much to formulate incorrect opinions regarding this shadow and, no doubt, has delayed its recognition as a forerunner of and a condition associated with a common form of bronchiectasis in children.

Singer and Graham [6] (1926) appeared to be the first in America to recognize the triangular shadow as a sign of atelectasis and bronchiectasis

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1. Polak, J. B.: Pleuritis mediastinitis, Acta radiol. **2**:461, 1923.

2. Schönfeld, H.: Beiträge zur Röntgendiagnostik pleuritischer Prozesse im Säuglings und Kindesalter, Monatschr. f. Kinderh. **39**:385, 1928. Stoloff, E. Gordon: The Chest in Children, Roentgenologically Considered, Ann. Roentgenol. **12**:1 (Nov.) 1930; New York, Paul B. Hoeber, Inc., 1930, p. 265. Tice, F.: Posterior Mediastinal Pleurisy, M. Clin. North America **5**:157, 1921.

3. Sagel, Jacob, and Rigler, Leo G.: Mediastinal Pleural Effusion, Am. J. Roentgenol. **24**:225, 1930.

4. Rigler, L. G., and Ericksen, L. G.: The Inferior Accessory Lobe of the Lung, Am. J. Roentgenol. **29**:384, 1933.

5. Wessler, H., and Jackes, L.: The Mediastinum, Clinical Roentgenology of Diseases of the Chest, Troy, N. Y., The Southworth Company, 1923, p. 404.

6. (*a*) Singer, J. J.: Diagnostic Pneumothorax, Ann. Clin. Med. **4**:907, 1926. (*b*) Singer, J. J., and Graham, E. A.: Roentgen-Ray Study of Bronchiectasis, Am. J. Roentgenol. **15**:54, 1926.

of the lower lobe. Many authors [7] have made the same observations, and recently an increasing number of physicians interested in the subject have learned to regard the triangular shadow as a sign of atelectasis and bronchiectasis. Richards [7d] recently stated that the triangular shadow was pathognomonic of these two conditions. Bezancon [8] believed bronchiectasis to be the primary condition and atelectasis an associated finding.

The observations in a large group of patients observed either at necropsy or over long periods, from infancy to the end of childhood in some cases, have been so uniform that apparently a common etiologic basis of a certain process is indicated by the triangular shadow. The purpose of this paper is to present a picture of the trends and variations in these cases, to bring out the correct interpretation of the triangular shadow, to demonstrate the successive steps in the production of a common form of bronchiectasis in children and to show the benefit of certain prophylactic procedures when started early and continued over long periods.

THE TRIANGULAR BASAL PULMONARY SHADOW

As seen in regular roentgenograms of the chest the shadow common to this group can best be described as a small well defined dense right triangle (fig. 1 *A*). The mesial border, or altitude, and the inferior border, or base, in the order named, are indistinguishable from the shadows of the spine and leaf of the diaphragm. The well defined lateral border, or hypotenuse, of the triangle extends from the hilus to a variable point on the diaphragm. The mesial portion of the shadow is partly obscured by the cardiac shadow because the heart, diaphragm

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7. (*a*) Singer and Micham.[6b] (*b*) Rist, E.; Jacob, P., and Irocuré, P.: Pleurésie médiastine et bronchiectasie, Ann. de méd. **21**:144, 1927. (*c*) Wallgren, A.: Der basale Dreieckschatten und seine diagnostische Bedeutung, Beitr. z. klin. d. Tuberk. **69**:641, 1928. (*d*) Richards, G. E.: The Interpretation of the Triangular Basal Shadows in Roentgenograms of the Chest, Am. J. Roentgenol. **30**:289, 1933. (*e*) Pinchin, A. J. S., and Morlock, H. V.: Atelectatic Bronchiectasis, Brit. M. J. **1**:12 (Jan. 4) 1930. (*f*) Jones, O. R., and Cournand, A.: The Shrunken Pulmonary Lobe with Chronic Bronchiectasis, Am. Rev. Tuberc. **28**:293 (Sept.) 1933. (*g*) Warner, W. P., and Graham, Duncan: Lobar Atelectasis as a Cause of Triangular Roentgen Shadows in Bronchiectasis, Arch. Int. Med. **52**:888 (Dec.) 1933.

8. Bezancon, F.; Weil, M. P.; Azoulay, R., and Bernard, E.: Forme sèche hémoptoïque de la dilatation des bronches, Presse méd. **32**:157, 1924.

and other adjacent structures are drawn toward the involved side. In lateral roentgenograms this density is seen as an isosceles triangle (fig. 1 *B*) with its apex at the hilus and its base on the diaphragm.

The following twenty cases with illustrations offer an explanation of the triangular density quite different from that of previously published reports.

REPORT OF CASES

CASES IN WHICH PATIENTS CAME TO NECROPSY AT AN EARLY AGE

CASE 1.—*Triangular shadow, with early bronchial dilatations following possible congenital atelectasis; necropsy.*

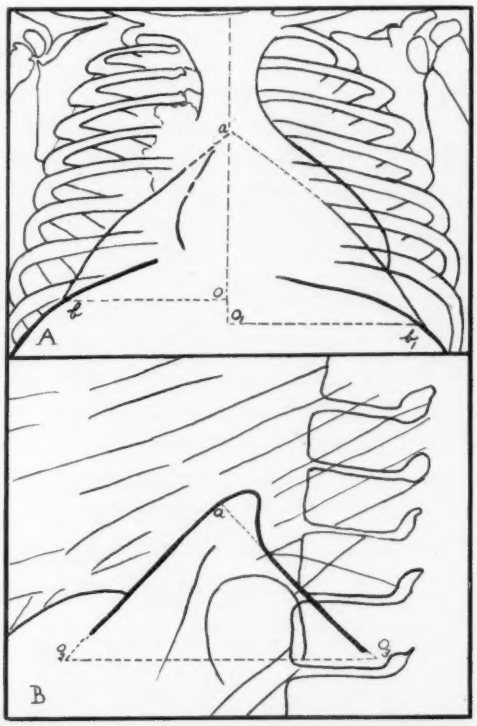

Fig. 1.—*A*, tracing illustrating the appearance of the triangular density as seen in the frontal view. In this instance the lesion is bilateral. The lesion on the right side appears as a right triangle (*aob*) in which most of the hypotenuse (*ab*) is a real line discernible in the roentgenogram. The altitude (*ao*) and the base (*bc*) are imaginary lines. On the left the triangle (ao_1b_1) is not so well defined, being overlaid by the cardiac shadow. The original roentgenograms are shown in figures 8*A* and 8*B*. *B*, tracing of the lateral right view, from the same case as *A*. In this view the lesion on the right side again shows as a triangle, but this time two sides ao_2 and ao_3, are real lines.

History: R. B., a full-term, white male infant, aged 3 months, was cyanotic and had a hoarse cry for the first three days of life. Throughout the first month there was a dry, weak cough, which once showed improvement for a couple of

weeks, followed by pronounced severity. On admission to the hospital, the respirations were jerky and rapid. There were paroxysms of severe coughing. The temperature was never elevated more than 1 degree. The cyanosis increased and the child died two days after admission.

Roentgen Findings: A roentgenogram of the chest (fig. 2 *A*) showed a dense triangular shadow in the base of the right lung and displacement of the heart to the right side with a faint network of shadows extending above and laterally for a short distance with increased translucency just above the triangular area of density.

Necropsy: The lower lobe of the right lung was collapsed to little more than half the size of the normal left lobe. The pleura showed no changes. No adhe-

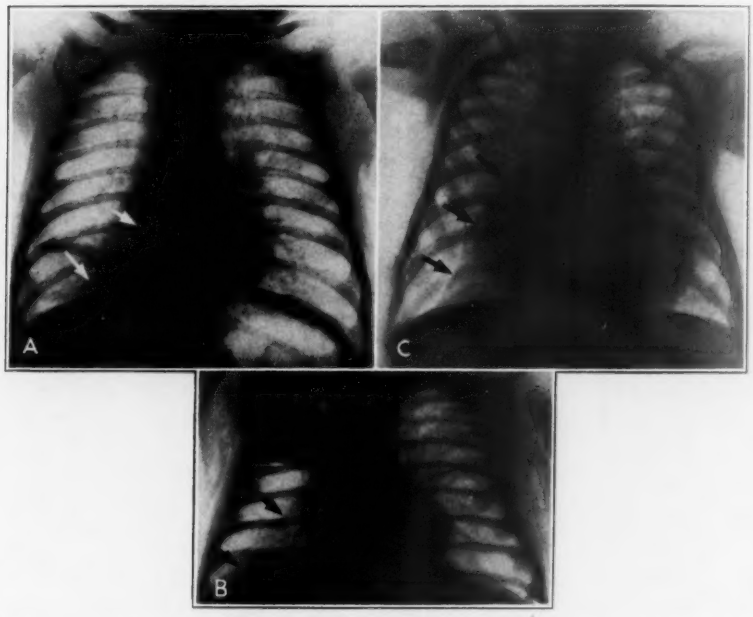

Fig. 2.—*A* (case 1, age 3 months), small triangular shadow at the base of the right lung. Necropsy revealed atelectasis of the lower lobe of that lung. *B* (case 2, age 11 days), faint triangular shadow at the base and dense area in the upper half of the right lung. Necropsy revealed partial atelectasis of the lower and upper lobes of that lung. *C* (case 3, age 5 weeks), small triangular cardiac shadow at the base of the right lung and enlarged cardiac shadow. Autopsy showed a collapsed lower lobe of that lung and a congenital cardiac lesion.

sions were found between the lower lobe of the right lung and the surrounding structures. The bronchi were filled with yellow necrotic matter. Slight dilatations, not noticeable except on closest scrutiny, were found in a few places. The pulmonary tissue was solid, dull and reddish blue. The upper lobes of the right lung were pale and greatly distended. On the surface a shallow furrow, composed of atelectatic pulmonary tissue, marked the location of a transverse fissure. Marked emphysema of other portions of the right lung was found. The left lung showed no abnormal changes, except slight emphysema of the anterior lappets.

The pathologic diagnosis based on the necropsy observations was that of acquired, almost complete, atelectasis of the lower lobe of the right lung with possible early bronchiectatic changes (fig. 18).

CASE 2.—*Triangular shadow with no evidence of bronchial dilatation at necropsy.*

History: T. S., a white female infant, aged 11 days, had had extensive blisters over the face, legs and body since 4 days of age. The child was breast fed, but did not take feedings well. A temperature of 104 F. had developed the day before admission to the hospital. Dry, coarse, sibilant râles were present over the entire chest posteriorly. Bronchophony and bronchial breathing were present in the right subscapular region. The child died forty-eight hours after admission.

Roentgen Findings: A dense triangular basal shadow appeared in the right cardiohepatic angle; there was displacement of the heart to the right. In the upper part of the right lung field there was a clouded area interpreted as a consolidation of the upper lobe of that lung (fig. 2 *B*).

Necropsy: There was partial atelectasis of the lower and upper lobes of the right lung with no evidence of bronchial dilatation. Small areas of bronchopneumonia were present in the left lung; these were considered terminal.

CASE 3.—*Small, sharply defined triangle at the base of the right lung; parabronchial infiltration and collapse of the alveoli at necropsy.* (Patient followed from the age of 5 weeks to 6½ months.)

History: In B. D., a colored girl, aged 5 weeks, who was undernourished, fever, dyspnea and cough had developed rather suddenly the day before admission to the hospital. There had been no previous illness. On admission the temperature was 105.6 F., and there was an expiratory grunt. A physical examination revealed: (1) a saddle-shaped nose with a marked mucopurulent discharge; (2) a red and injected pharynx; (3) marked cervical adenopathy; (4) marked retraction of the lower part of the thorax and prominence of the sternum during inspiration; (5) a bilaterally enlarged heart, and (6) dulness and crackling râles over the base of the right lung posteriorly. Bronchopneumonia was thought to be present in the base of the right lung. The temperature dropped on the second day, but remained slightly elevated for four days. Then it fluctuated between 96.4 and 99 F. for two weeks, at which time the patient left the hospital. At home the child did poorly; her temperature hovered around 99 F. After an interval of one month she returned to the hospital. There was considerable coughing, especially on awakening, but the temperature remained about normal. The Wassermann reaction was 4 plus. Four days after returning to the hospital, the child became cyanotic and died suddenly.

Roentgen Findings: A roentgenogram of the chest on admission showed bilateral mottling of the soft tissues of the inner third of both pulmonary fields. A triangular area of homogeneous density was present in the base of the right lung, as shown in figure 2 *C*. A roentgenogram made at the time of the second admission showed the appearance to be unchanged.

Necropsy: This revealed a consolidated lower lobe of the right lung and areas of bronchopneumonia in other lobes, together with a congenital cardiac lesion. There were no pleural adhesions, no free fluid and no white pneumonia. Sections of tissue from the lower lobe failed to show abnormalities of the bronchial wall other than a fibroblastic reaction in the surrounding tissue. The alveolar walls were close together and appeared as though it would be possible to reexpand them.

CASE 4.—*Triangular shadow; no bronchiectasis at necropsy.*

History: J. B., a white Norwegian boy, was admitted to the hospital at the age of 30 months. Five months previously, following impetigo and infection with Vincent's organism, there had been loss of weight and emaciation which had increased over that period. Physical examination on admission revealed a temperature of 105 F., a papular rash over the body, redness of the hands and feet and a geographic tongue. There were also signs of consolidation in the lower lobe of the right lung. The examining physician made the following note: "There is bronchophony as well as bronchial breathing over the right lung posteriorly without much impaired resonance. In the absence of fever, I believe this is a

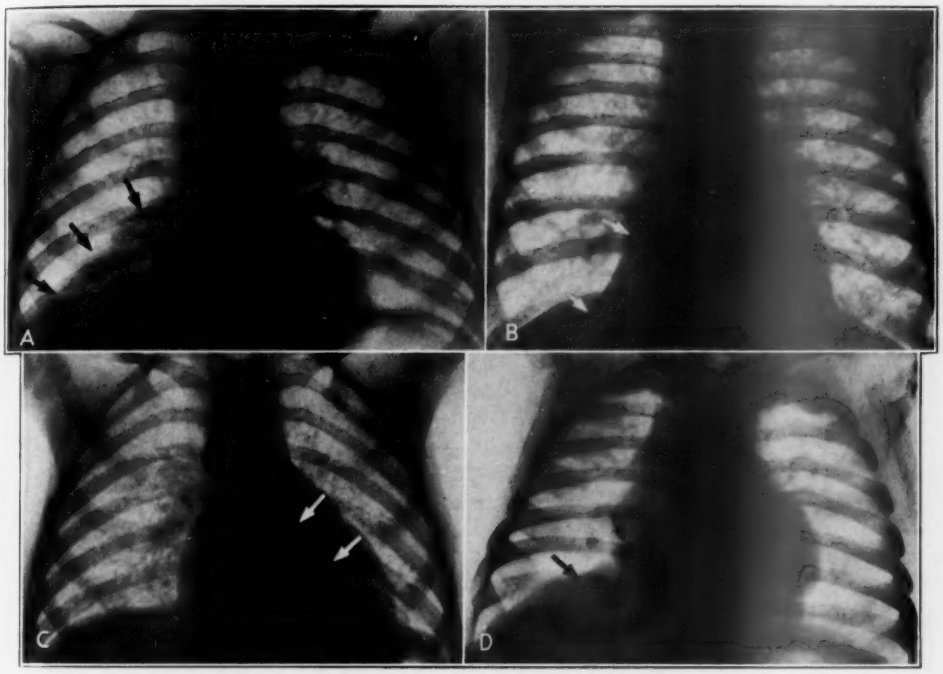

Fig. 3.—*A* (case 4, age 2 years and 6 months), triangular shadow at the base of the right lung. Necropsy showed a collapsed lower lobe and bronchi filled with thick exudate. *B* (case 5, age 9 months), sharply outlined small triangle at the base of the right lung. An anomaly was expected, but necropsy revealed a collapse of the lower lobe of that lung, which accounted for the shadow. *C* (case 6, age 20 months), triangular shadow at the base of the left lung following a subphrenic infection. Necropsy revealed adhesions and exudate about the solid lower lobe of that lung. *D* (case 7, age 15 months), triangular shadow at the base of the right lung. This was thought to be of tuberculous origin. Repeated tuberculin tests were negative.

collapsed lobe due to a plugged bronchus." Sinking spells increased over a period of one month, and cyanosis developed. The patient finally became unable to expectorate; the throat filled with secretion, and four days after the development of this condition death occurred. A diagnosis of acrodynia had been made.

Roentgen Findings: A roentgenogram (fig. 3*A*) showed the characteristic triangular shadow at the base of the right lung with corresponding displacement of the heart to the right.

Necropsy: There was marked tracheobronchitis, with complete collapse of the lower lobe of the right lung, but no gross evidence of bronchiectasis, pleural fluid or adhesions (fig. 15). Photomicrographs are shown in figures 16 and 17.

CASE 5.—*Triangular shadow with asymmetrical chest and decreased expansion on the affected side; necropsy.* (Patient followed from the age of 6 to 9 months.)

History: L. M., a white male infant, aged 6 months, had been born after seven months' gestation and had failed to gain weight satisfactorily. There was a history of shortness of breath for two months. On admission to the hospital he breathed with difficulty, and there was a mild cough. The chest was asymmetrical, and during inspiration there appeared to be very little expansion. There was decidedly less expansion on the right than on the left side, and the right lung was hyperresonant. On admission, the temperature was 103.8 F.; after twenty-four hours it fell to subnormal, and over a period of twenty-five days it varied from 94.4 to 98.6 F. There was a thick purulent discharge from the left nostril, and after a few days severe paroxysms of coughing developed. The Pirquet reaction was negative. The child failed to gain; dyspnea became more pronounced, and death occurred three months after admission.

Roentgen Findings: Roentgenograms made at different intervals showed a triangular shadow at the base of the right lung, as shown in figure 3*B*. There were also mottled densities in the region of both hili.

Necropsy: The lower lobe of the right lung was found to be unusually small and crowded into the right postcardiac sulcus. Histologic section showed extensive collapse, but no bronchial dilatations were evident.

CASE 6.—*Triangular shadow in presence of subphrenic infection; pleural exudate about collapsed lower lobe at necropsy.* (Patient followed for three days.)

History: M. C., a white female infant, was brought to the hospital when 20 months of age. On admission the temperature was 103 F. There were boardlike rigidity and bulging of the hypogastrium accompanied by severe abdominal pain. The child was believed to have a localized peritonitis. Many râles were also present over the base of the left lung, and a pulmonary abscess was thought to exist. Laparotomy was performed, and many pockets of pus were drained, but the child died twenty-four hours later.

Roentgen Findings: A roentgenogram (fig. 3*C*) showed a triangular density superimposed on the cardiac shadow at the base of the left lung, with displacement of the heart to the left.

Necropsy: Pockets of pus were found, one of which communicated with the under surface of the left leaf of the diaphragm. There was a small quantity of thick exudate at the base of the left pleural cavity. A marked decrease was noted in the size of the lower lobe at the point of the triangular shadow on the roentgenogram. There had been a perforating ulcer of the posterior wall of the stomach 3 mm. in diameter. On section of the lower lobe the bronchi were found filled with thick exudate. There was no evidence of bronchial dilatations or of a break in the bronchial walls.

CASE 7.—*Triangular shadow in infant of mother with open tuberculosis; fatal termination.* (Patient followed from the age of 10 to 15 months.)

History: J. C., a poorly developed, white male infant was brought to the hospital. The child had recently lost weight, but did not look ill. As the mother

was known to have active pulmonary tuberculosis, the family became alarmed over the loss in weight and the persistent cough. Physical examination revealed many moist râles at the bases of both lungs; these were more pronounced on the left side. Numerous tuberculin tests were negative. The leukocyte count reached 15,200 on one occasion. The infant was observed over a period of five months, during which time weight was gained, only to be lost later. The child finally was taken home and died in a few days. No necropsy was performed.

Roentgen Findings: Several roentgenograms showed the presence of a triangular shadow at the base of the right lung with displacement of the heart and trachea to the involved side. Figure 3 *D* shows the triangular shadow which fluctuated at times during the period of observation but never disappeared. When the triangular shadow was not so pronounced, the heart assumed a position more nearly normal.

REPRESENTATIVE CASES IN WHICH THE TRIANGULAR DENSITY FLUCTUATED AND ULTIMATELY DISAPPEARED

CASE 8.—*Triangular densities at the bases of both lungs for eight years; clearing without development of bronchiectasis following postural drainage.* (Patient followed from the age of 15 months to 13 years and 6 months.)

History: L. G., a well nourished, white female infant, was admitted to the hospital when 15 months of age. On admission the temperature was 104 F.; this subsided in thirty-six hours, ranging after that from 98.6 to 99.2 F. Physical examination revealed rapid respirations, grunting, a dry irritative cough, a watery nasal discharge and double otitis media. Dulness and tubular breathing were evident at the base of the right lung, with less marked dulness posteriorly from the spine to the axillary line. Tubular breathing, bronchophony and râles were present on the left side. The leukocyte count reached 16,300. The tuberculin and Wassermann reactions were negative. Postural drainage was begun, and after five weeks the child left the hospital slightly improved. The cough and periods of slightly elevated temperature persisted. The patient was an almost constant visitor at dispensaries and later spent a year in a sanatorium for tuberculous patients. Postural drainage seemed to affect the size and density of the shadow, but the symptoms persisted from the age of 15 months until the child was 8 years of age. The child is now 13½ years old; no symptoms have been present in the past five and a half years, and at present the development is normal.

Roentgen Findings: A roentgenographic examination showed partial obliterative triangular shadows at the bases of both lungs on either side of the spine (fig. 4 *A*). These shadows fluctuated over a period of years and finally cleared. A recent examination, twelve years after the onset, showed moderate scarring of both hili but no signs of bronchiectasis (fig. 4 *B*).

CASE 9.—*Triangular shadows which fluctuated following postural drainage; development of bronchiectasis in five years.* (Patient followed from the age of 8 months to 6½ years.)

History: C. S., a frail, white female infant was brought to the dispensary when 8 months of age. On admission to the hospital there was a temperature of 104 F., accompanied by grunting respirations and cyanosis. The child was thought to have pneumonia. The high temperature subsided in twenty-four hours, but a loose cough and shortness of breath persisted and became almost constant symptoms. The child improved and left the hospital, but in the succeeding four years was frequently a patient at the dispensary and was readmitted

to the hospital six times. At various times there have been discharge from the ears, dental caries and hypertrophied tonsils. The urine has shown large amounts of albumin with an occasional granular and hyaline cast. Postural drainage was instituted early, and a recent examination showed a gain in weight and apparently great improvement.

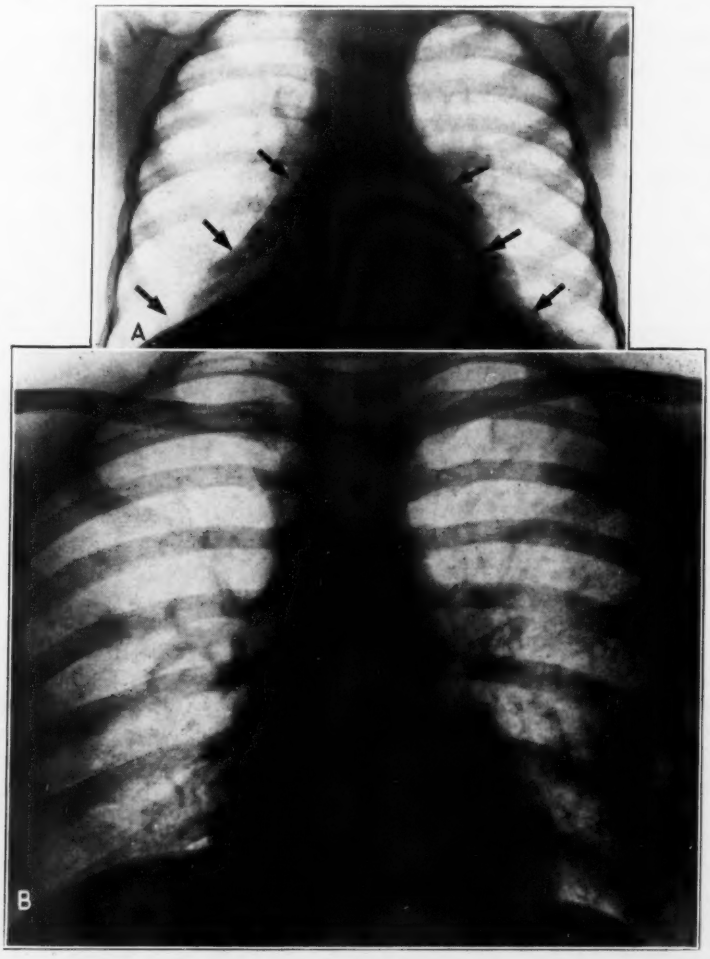

Fig. 4.—*A* (case 8, age 15 months), bilateral triangular shadow with little cardiac displacement. Note the gap in the lateral margin of the left triangle due to incomplete collapse. *B* (case 8, age 13 years and 6 months), appearance twelve years after the roentgenogram in figure 4 *A* was taken. Note the absence of triangles. The patient was well for five years.

Roentgen Findings: A roentgenogram of the chest taken at the first examination showed a faint triangular shadow at the base of the right lung. Many roentgenograms of the chest have been made during the five years this patient

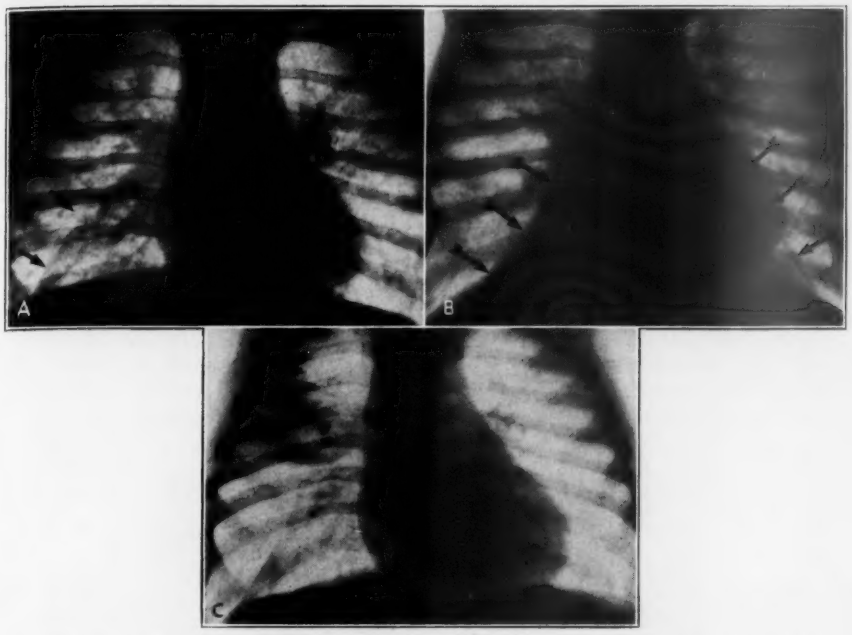

Fig. 5.—*A* (case 9, age 3 years), moderately large triangular shadow of slightly increased density at the base of the right lung. Accentuated markings superimposed on the left cardiac shadow. *B* (case 9, age 3 years), appearance two weeks later. Note the unusual decrease in size and increase in density of the triangle at the base; also the increase in density of the triangle at the base of the left lung. *C* (case 9, age 3 years and 6 months), partial clouding of the lower half of the upper lobe of the right lung. This occurred during the observation of the triangular shadow at the base of that lung. Later the shadow disappeared. This is interpreted as a spilling over of secretions into an adjacent lobe.

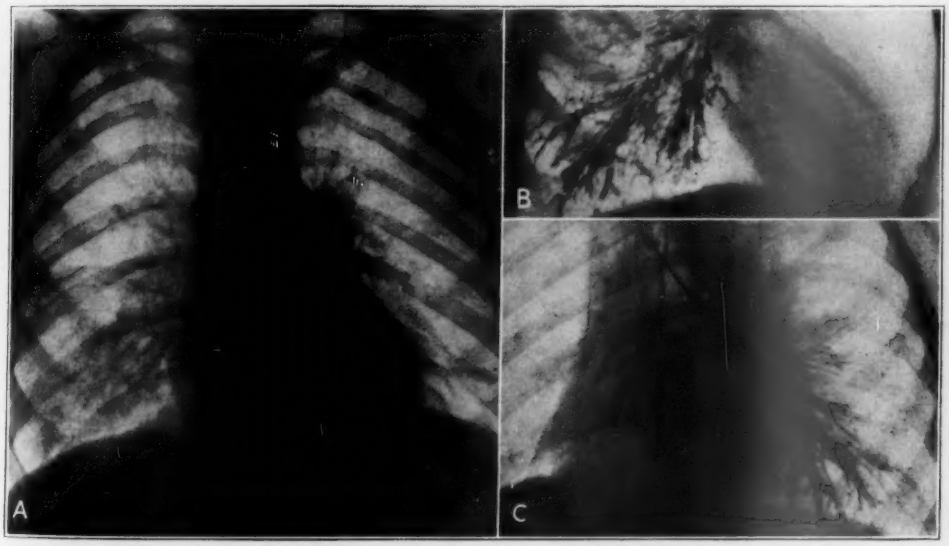

Fig. 6.—*A* (case 9, age 7 years), appearance four years later, after postural drainage. Note the disappearance of the triangle at the base of the right lung and the increased inflation of the triangle at the base of the left lung. *B* and *C* (case 9, age 7 years), lateral and oblique views after injection of iodized poppy-seed oil 40 per cent showing the absence of marked bronchial dilatations.

has been under observation. During this time there has been marked fluctuation in the size and density of the triangle which first appeared on the right and later on the left side. Following prolonged postural drainage the shadows have been typically less dense when large (fig. 5 *A*) and more dense when small (fig. 5 *B*). A recent examination following an injection of iodized poppy-seed oil 40 per cent showed possible evidence of tubular dilatations (fig. 6 *B* and *C*). There is scarring over the lower half of the pulmonary field (fig. 6 *A*). The triangle on the right side has entirely disappeared, and on the left there is an increased amount of air-containing tissue. However, there is still the suggestion of a triangular density on the left side, but no definite signs of the development of bronchiectasis.

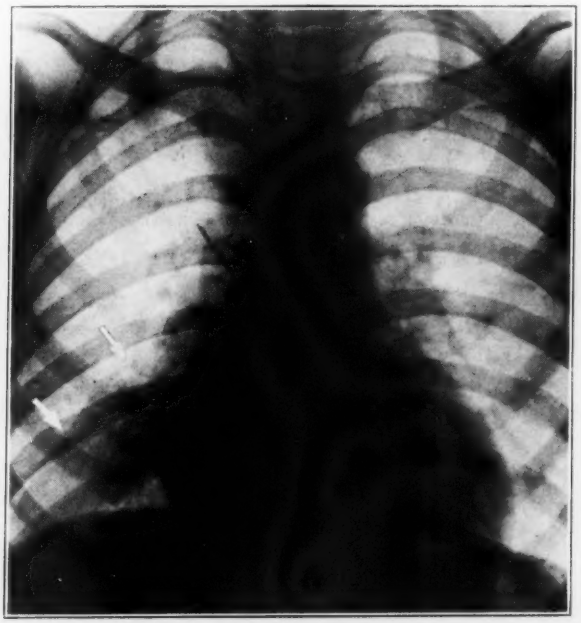

Fig. 7 (case 10, age 11 years).—Faint large triangular shadow at the base of the right lung. The foreign body, a doll's eye, has been present in the main bronchus of the lower lobe of that lung for twenty-two months. There is no evidence of bronchiectasis.

CASE 10.—*Triangular shadow following partial obstruction of the main bronchus by a foreign body; no bronchiectasis.* (Patient followed from the age of 9 to 11 years.)

History: M. S., a girl aged 9 years, twenty-two months before admission to the hospital had swallowed a doll's eye, consisting of a bead with a wire attached to it. There was a history of slightly elevated temperature at times and a more or less constant cough.

Roentgen Findings: A roentgenogram (fig. 7) revealed a large, slightly dense triangle in the base of the right lung below the foreign body. Following the removal of the object, this triangle cleared within sixty days, leaving practically no visible markings; there were no clinical symptoms.

CASE 11.—*Triangular shadows at the bases of both lungs; one clearing and one persisting with subsequent development of bronchiectasis.* (Patient followed from the age of 15 months to 13 years and 6 months.)

History: M. N., a white female infant was brought to the hospital when 15 months of age. On admission the temperature was 106 F. and there was a hacking cough. The temperature subsided at the end of twenty-four hours and subsequently was not higher than 101 F. Up to that time the child had been in good health. Physical examination showed a slight right-sided bulging of the chest, limited expansion and diminished breath sounds on the left side. The leukocyte count was 28,650. It was thought that empyema was present on the left side, but thoracentesis revealed only a small amount of thick mucopus and blood. The Pirquet test for tuberculosis was negative. A cough, for the most part nonproductive, persisted. After two months the patient left the hospital unimproved. Within the succeeding twelve years, she has been readmitted to the hospital

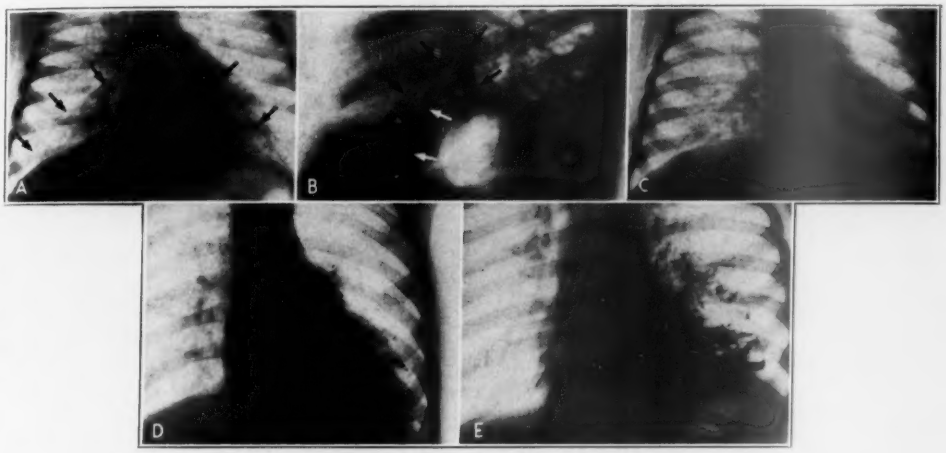

Fig. 8.—*A* (case 11, age 17 months), bilateral triangles at the bases. Note the greater density on the left with displacement of the heart to the left. The appearance is almost identical with that of case 8 (fig. 4*A*). *B* (case 11, age 20 months), lateral view. The black arrows indicate the outline of the collapsed lower lobe, which is flattened out on the elevated leaf of the diaphragm. The white arrows indicate the posterior margin on the fold of pulmonary tissue. Note the somewhat linear shadow which in many cases has led to a mistaken diagnosis of interlobar pleurisy. Hyperaeration of the upper lobe and gas in the stomach and bowels mitigate the density of the posterior portion of the shadow of the lower lobe. *C* (case 11, age 3 years and 1 month), appearance eighteen months later. Note the increase in displacement of the heart and the increase in density on the left. Note also the disappearance of the triangle on the right. *D* (case 11, age 10 years), appearance eight years and six months later. Note the triangular shadow at the base of the left lung, with a hazy lateral border. Iodized poppy-seed oil 40 per cent has shown a saccular bronchiectasis in the lower lobe of that lung. *E* (case 11, age 13 years and 6 months), appearance twelve years after the roentgenogram in figure 8*A* was taken. Partial reexpansion of the lower lobe of the left lung. Iodized poppy-seed oil 40 per cent is present in the large sacculations.

eleven times. On each occasion the complaint has been similar, that is, persistent cough and at times a low grade fever. The only change in the cough from the beginning is that it has become more moist and productive.

Roentgen Findings: Many roentgenograms have been made. At first, there were well defined triangular shadows in the mesial portions of the bases of both lungs (fig. 8*A*). Later the right side cleared, but the triangle on the left side persisted, and within eighteen months after the onset, there were signs of bronchiectasis on the left side (fig. 8*C*). Figure 8*D*, a roentgenogram taken when the patient was 10 years of age, shows bronchiectasis at a later stage with loss of

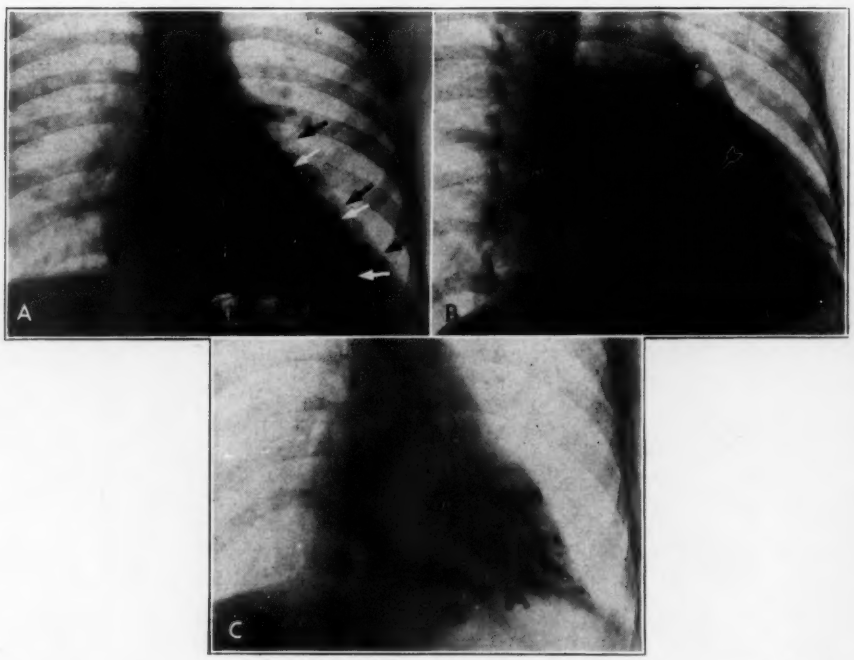

Fig. 9.—*A* (case 12, age 5 years), large triangular shadow with the hypotenuse parallel to the left cardiac border. The black arrows show the hypotenuse, and the white arrows show the cardiac border. *B* (case 12, age 5 years), right view showing the triangular shadow flattened out on the diaphragm. *C* (case 12, age 7 years), same case as that shown in figure 9*A*. Iodized poppy-seed oil 40 per cent brings out bronchial dilatations in the region of the previously noted triangular shadow.

the well defined lateral border of the triangle. The roentgenogram in figure 8*E*, taken at 13 years and 6 months following injections of iodized poppy-seed oil 40 per cent, reveals a well developed saccular bronchiectasis in the zone originally occupied by the triangular shadow at the base of the left lung. Figure 8*B*, a lateral view, shows the marked retraction of the lower lobe forming an isosceles triangle.

REPRESENTATAVE CASES IN WHICH THE TRIANGULAR SHADOW REMAINED FIXED AND BRONCHIECTASIS DEVELOPED

CASE 12.—*Large triangular shadow with saccular bronchiectasis in which the lower lobe was flattened out on the diaphragm.* (Patient followed for four years from the age of 5 to 9 years.)

History: M. S., a poorly nourished, pale, white girl, was admitted to the hospital when 5 years of age. There was a history of the presence of a moist, productive cough for four years following an acute pulmonary illness thought to have been pneumonia. There was no history of other antecedent diseases. At the time of the first visit and for four years afterward there were slightly enlarged cervical glands, a loose free cough and diminished breath and voice sounds posteriorly in the base of the left lung. Many moist râles, inspiratory in character, have been present. By means of inverted posture, from ½ and 1 ounce (14.17 and 28.35 Gm.) of nonfoul-smelling pus has often been secured. There has been slight leukocytosis. The sinuses are clear; the tuberculin test is negative, and the urine is normal. At first a pulmonary abscess was thought to be present, but later a diagnosis of bronchiectasis was made.

Roentgen Findings: Roentgenograms of the chest taken on admission showed a triangular shaped density in the base of the left lung. The lateral border of this density was parallel to the cardiac border (fig. 9 *A*). Different silhouettes of this shadow were obtained which revealed its flattened position on the diaphragm (fig. 9 *B*). The injection of an opaque oil brought out in the roentgenograms the outline of the bronchial trunks extending into the triangular shadow, and showed several moderately large bronchial dilatations (fig. 9 *C*).

CASE 13.—*Triangular shadow following measles and pneumonia; tubular bronchiectasis.* (Patient followed from the age of 18 months to 7 years.)

History: W. K., a white female infant, was admitted to the hospital at 18 months of age. There was a history of measles, pertussis and pneumonia closely following each other; then a cough developed. There was profuse expectoration, especially in the morning, when as much as ½ ounce (14.175 Gm.) of pus was frequently raised. In the fourth year of life a discharge developed in both ears, and there has been a nearly continuous discharge since. An examination made at 7 years of age showed impaired resonance over the base of the left lung. After the patient coughed and raised half an ounce of pus there were bronchial breathing and bronchophony. Slight clubbing of the fingers has occurred.

Roentgen Findings: Roentgenograms taken at various intervals revealed a triangular shadow superimposed on the left cardiac shadow. This shadow fluctuated but little and had a striped density as shown in figure 10 *A*. The injection of iodized poppy-seed oil 40 per cent revealed tubular bronchiectasis of the lower lobe of the left lung, accounting for this striped appearance of the density (fig. 10 *B*).

CASE 14.—*Triangular shadow; saccular bronchiectasis following bronchitis.* (Patient followed from the age of 18 months to 8 years.)

History: J. J., a white male infant, was brought to the hospital at the age of 18 months. A severe and productive cough had been present for six months. There was a history of an acute onset with a chill and a high temperature which lasted for three days. The mother stated that when the child was 6 months of age, bronchitis had developed, and that the bronchial symptoms had never entirely disappeared. Following measles the cough had become more productive, and it

was thought that there might be a pulmonary abscess. On admission, the child appeared well developed and well nourished. Over several months the temperature varied only from 99 to 99.8 F., and the cough persisted, accompanied by many harsh, moist râles. At 7 years of age, there was not only flatness, but accentuation of the breath sounds over the base of the right lung.

Roentgen Findings: Roentgenograms taken when the child was first seen showed a triangle of fluid-like density on the right side. This density has persisted without much variation. Figure 10*C* shows a triangular density at the base of the right lung. Figure 10*D* shows marked sacculations brought out by the injection of iodized poppy-seed oil 40 per cent. On the basis of these roentgen

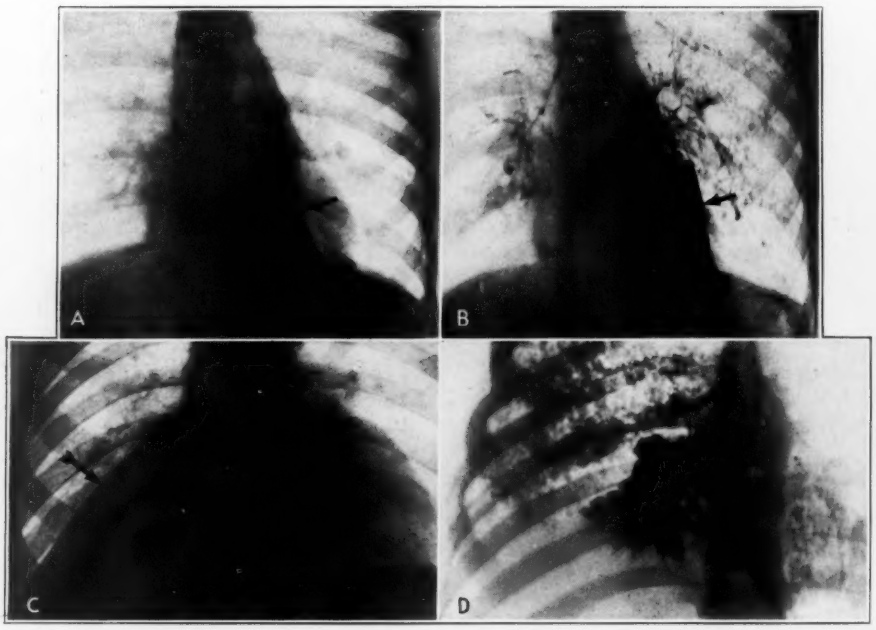

Fig. 10.—*A* (case 13, age 7 years), triangular shadow with striped pattern of density at the base of the left lung. Partial collapse of the lower lobe of that lung. *B* (case 13, age 7 years), appearance after the injection of iodized poppy-seed oil 40 per cent. Note the marked tubular dilatations within the triangular shadow. *C* (case 14, age 8 years), dense triangular shadow at the base of the right lung with smooth linear lateral border and oval areas of decreased density. *D* (case 14, age 8 years), the injection of iodized poppy-seed oil 40 per cent reveals a marked saccular bronchiectasis at the site of the triangular shadow.

findings, together with the clinical history, it appeared that bronchiectasis developed at some time between the age of 6 and 18 months and had persisted.

Case 15.—*Triangular shadow following complete obstruction of a bronchus by a foreign body; loss of lateral border of triangle and development of small bronchial dilatations.*

History: F. C., a white boy, aged 3 years, had aspirated a scarf-pin nine months before admission to the hospital. The child was brought to the hospital

because of a low-grade temperature and a moist productive cough which had been present for nine months. There was a history of a slightly elevated temperature at times. After the removal of the pin the bronchial symptoms continued, and there was marked expectoration, particularly in the morning.

Roentgen Findings: The pin was shown partially blocking the lumen to the lower lobe of the right lung. A triangular shadow was present, similar in outline to that noted in the later stages of saccular bronchiectasis in which the lateral border of the triangle is not clearly defined (fig. 11 *A*).

CASE 16.—*Triangular shadow thought to be encapsulated fluid; thoracentesis unsuccessful; atelectatic lower lobe.*

History: F. O., a poorly nourished white girl, aged 8 years, had a history of restlessness, weakness and cough for six weeks preceding admission to the hospital. In the beginning there had been frequent chills with fever and a nonproductive cough. Nausea and vomiting had occurred for the first two days. Because of the bronchial symptoms, a diagnosis of bronchitis had been made. On

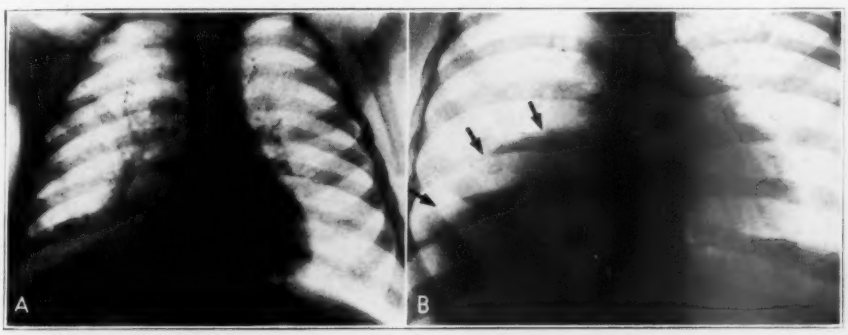

Fig. 11.—*A* (case 15, age 3 years), triangular shadow with a hazy lateral border at the base of the right lung following the occlusion of a bronchus by a foreign body which had been present for nine months. Note the similarity to advanced cases of bronchiectasis following an infection of the upper respiratory tract. *B* (case 16, age 8 years), early homogeneous triangular shadow in the base of the right lung with a convex lateral border. The heart is displaced toward the involved side.

admission to the hospital, the cheeks were flushed and the lips red and dry; there was an almost constant short dry cough. The history previous to this was that of a normal child. Physical examination showed a symmetrical thorax with good resonance over the left lung and the upper portion of the right lung. There were flatness and nearly complete absence of breath sounds posteriorly over the base of the right lung. The leukocyte count was 16,800, with 71 per cent polymorphonuclears. The Pirquet reaction was positive. The sputum was negative for tubercle bacilli. Empyema was thought to be present, but thoracentesis twice attempted on the right side failed to yield fluid. Varicella developed while the child was in the hospital, and she remained there for four months. During that time, the temperature varied from subnormal to slightly elevated, but only on rare occasions did it reach more than 100 F.

Six weeks after leaving the hospital the patient returned because the cough had not disappeared. Physical examination revealed an unchanged condition of the

right lung. At this time the child remained in the hospital for two months, but the cough persisted. Little gain in weight was recorded, and the patient was discharged unimproved.

Roentgen Findings: A series of roentgenograms taken while the child has been under observation show a triangular shadow at the base of the right lung, which has scarcely fluctuated and is constantly present (fig. 11 *B*).

CASE 17.—*Triangular shadow which disappeared and later reappeared; bronchiectasis present twelve years later.* (Patient followed from the age of 7 to 19 years.)

History: H. L., a white girl, came to the hospital when 7 years of age. Four years previously the patient had had an illness characterized by a high fever followed by weakness and a persistent cough. Following this there had been a cough and periods of rise in temperature in the winter months of each year. On admission, the child was undernourished and pale; paroxysms of coughing produced some expectoration. Physical examination revealed hyperresonance in the upper part of the pulmonary field. There were hyperresonance, diminished breathing, bronchophony and a tubular quality to breath sounds heard low between the

Fig. 12.—*A* (case 17, age 7 years), smooth homogeneous triangular shadow at the base of the left lung. Atelectasis is apparently complete. Because of the water-like smoothness, many conditions of this kind have been interpreted as fluid-containing pockets outside of the lung. *B* (case 17, age 7 years), appearance one month later. Note the loss of the triangular shadow. *C* (case 17, age 7 years), appearance two months later. Note the return of the smooth homogeneous triangular shadow.

scapula and the spine over the left lung. An area of dulness was thought to be present posteriorly at the base of the right lung. The leukocyte count was 7,600, and the sputum was negative for tubercle bacilli. There were a number of carious teeth; the tonsils were enlarged, and there was general cervical adenopathy.

Roentgen Findings: Roentgen examination revealed a smooth, dense triangular shadow partly obscured by the left cardiac shadow. The heart was displaced to the left (fig. 12 *A*). A second examination, one month later, showed the complete disappearance of the triangular area of density with the return of the heart to its normal position (fig. 12 *B*). A third examination, a few weeks later, showed the recurrence of the triangular area of density (fig. 12 *C*).

This patient was lost track of (over age) until recently, when it was discovered that a diagnosis of bronchiectasis had been made. All the clinical and roentgenologic signs of bronchiectasis are present, and, on inverted posture, there is profuse drainage.

CASE 18.—*Triangular shadow following a general anesthetic for removal of tonsils and adenoids; development of bronchiectasis.*

History: C. P., a white boy, came to the outpatient department when 8 years of age, showing mouth breathing, hypertrophied tonsils and adenoids, but no cough or expectoration. The tonsils and adenoids were removed under general anesthesia, and after a few days the patient went home. A month later, he returned with the history of an almost constant cough with profuse expectoration in the morning. This condition had been present ever since the operation for removal of the tonsils and adenoids. Inspection showed the tonsillar pillars to be red, and there was some drooping of the soft palate. The temperature varied from 97.2 to 99.4 F.

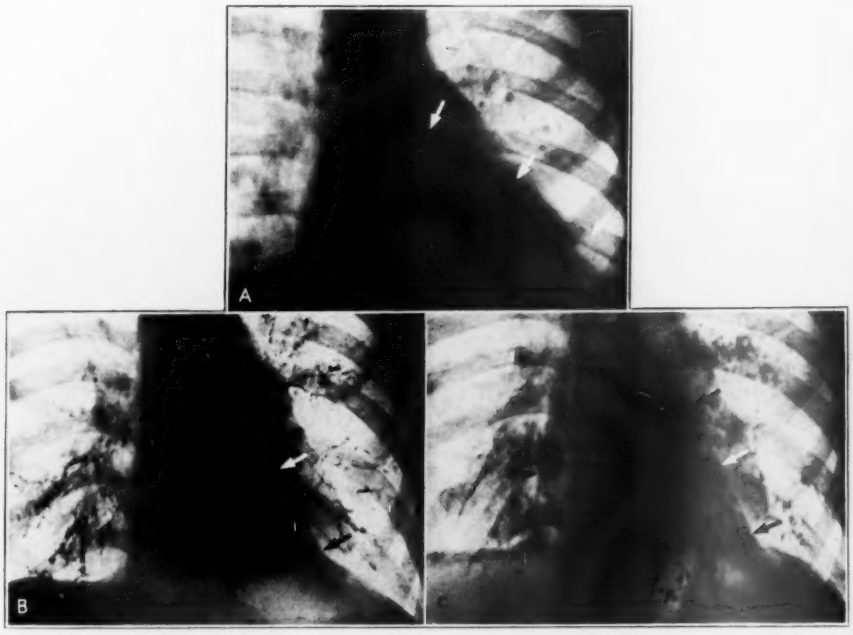

Fig. 13.—*A* (case 18, age 8 years), large triangular shadow at the base of the left lung. This was noted six months after ether anesthesia. *B* (case 19, age 10 years), following the injection of iodized poppy-seed oil 40 per cent the small triangle at the base of the left lung shows many sacculations. Note that the cardiac shadow is not visible to the right of the spine. *C* (case 19, age 10 years), this roentgenogram was taken with the patient in the same position thirty minutes later, following postural drainage. Note the minimum enlargement of the triangular shadow. The heart is now visible to the right of the spine. These changes are characteristic of rapidly developing bronchiectasis.

There were numerous subcrepitant râles in the left lung, posterior to and at the right of the scapula.

Roentgen Findings: Roentgenograms taken at the time of the second admission to the hospital revealed a large triangular shadow obscured by the cardiac shadow at the base of the lobe of the left lung (fig. 13 *A*). Subsequent roentgen examinations have shown slight change in the size or density of this shadow. Postural

drainage likewise has had slight effect on the character of the triangle. The injection of iodized poppy-seed oil 40 per cent recently revealed moderate bronchial dilatation.

This is an illustration of the early stage of bronchiectasis in an older child, in which the triangular shadow does not fluctuate but shows homogeneous density extending to the costophrenic angle.

CASE 19.—*Small dense triangular shadow at base of left lung with saccular bronchiectasis.* (Patient followed from the age of 9 to 10 years.)

History: E. D., a white boy, came to the hospital at 9 years of age. He had had perfect health until the year before, when an acute pulmonary infection occurred which was diagnosed as pneumonia. There was an initial temperature

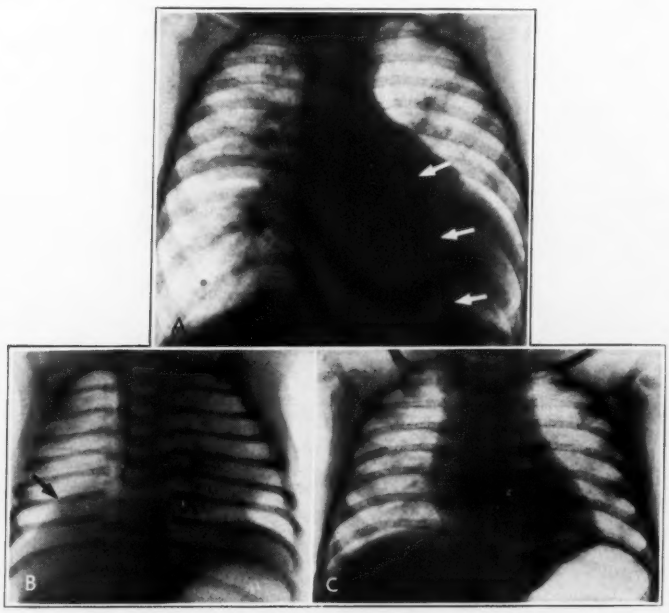

Fig. 14.—*A* (case 20, age 20 months), dense triangular shadow posterior to the enlarged cardiac shadow at the base of the left lung with displacement of the heart to the left. Compression atelectasis was thought to exist. *B* (J. B., case 21, age 6 hours), triangular shadow at the base of the right lung six hours after birth. There were grunting whistling respirations which cleared within a week. Congenital atelectasis accounts for the small size of the lobe. *C* (J. B., case 21, age 4 months), note the complete closing and disappearance of the triangle. In none of these cases did the collapse last long enough to cause bronchiectasis.

of 105 F. which subsided completely within thirty-six hours. Following that there was a slightly subnormal or moderately elevated temperature. For eight months there had been paroxysms of cough with considerable expectoration of thick mucus-like pus.

Roentgen Findings: Roentgenograms of the chest revealed a well defined triangular shadow at the base of the left lung with displacement of the heart to the involved side. Roentgenograms made after iodized poppy-seed oil 40 per cent

was injected into the triangular area revealed a moderate saccular bronchiectasis (fig. 13 *B*). Figure 13 *C* shows a minimum of expansion and slight return of the cardiac shadow to the midline after postural drainage.

REPRESENTATIVE TRIANGULAR SHADOW DUE TO COMPRESSION ATELECTASIS

CASE 20.—*Triangular shadow associated with congenital enlargement of the heart.*

History: M. T., a white female infant, brought to the hospital when approximately 20 months of age, appeared well developed, but had been abandoned in early infancy, and a satisfactory previous history was not obtainable. On admission cough and labored respirations were noted. Physical examination revealed tubular breathing posteriorly at the base of the left lung and the presence of a congenitally enlarged heart. The physician who examined the child the day before her admission to the hospital had made a tentative diagnosis of asthmatic bronchitis. At the hospital a diagnosis of congenital enlargement of the heart was made. During observation over five months in the hospital the temperature ranged from 99 to 100 F., and there were occasional paroxysms of cough, at times very moist.

Roentgen Findings: Several roentgenograms have shown the persistence of a dense, triangular shadow superimposed on the heart which is markedly enlarged and displaced to the left (fig. 14 *A*).

HISTORY OF CASES

The history common to the majority of cases is that of an acute pulmonary process at the onset. Elevation of temperature, which was frequently preceded by a chill, reached from 103 to 105 F. The physical findings approached those of lobar pneumonia. There was usually an early decrease of breath sounds near the base of the involved side. Moist râles were frequently heard. By the end of forty-eight hours the temperature usually dropped, often suddenly, and from that time on was seldom elevated more than 1 degree. It was sometimes subnormal. This slight fluctuation above or below normal continued for months during convalescence while the child was at home. There was a frequent hacking cough or practically no pulmonary symptoms for several weeks. Eventually the cough became moist, and expectoration was more frequent, especially after a period of sleep. Because of this moist, often severe cough, the child was likely to be returned to the hospital or to the care of a physician. If roentgenograms of the chest were made early, the triangular shadow was present, but often no dilatations of the bronchi were seen. In many instances, small dilatations were already formed by the time the patient returned. The latter seems to be especially true in children from 3 to 13 years of age. While younger children and infants showed the same changes, they were more apt to swallow secretions instead of expectorating. They did not fare as well as older children. Acute exacerbations of fever were more common. Diarrhea or infections of the upper respiratory tract were more

likely to be the cause of a return to the physician or the hospital. Infants in whom the triangular shadow was constantly present in the first year of life invariably died. In the second year, the majority of this group survived and in practically all untreated patients, bronchiectasis developed later. In many instances the chest was full of coarse crackling râles long before sacculations or even tubular dilatations became evident on the roentgenogram. Consequently bronchiectasis has been diagnosed clinically in cases that later completely cleared (case 8), but in which early signs were in every way parallel to those of other cases (case 11) in which bronchiectasis developed later.

Often the pulmonary symptoms, after the acute onset, were so mild that they were overshadowed almost completely by an infection of the upper respiratory tract. If an operation was anticipated, a preoperative roentgenogram of the chest sometimes revealed the triangular area of density, or the child vomited pus following the anesthesia, indicating the presence of the process. While infections of the upper respiratory tract frequently antedated the acute pulmonary condition, they were apparently later fed and maintained by the coughed-up infective secretions.

OBSERVATIONS AT DIFFERENT PERIODS

The character of the density of the triangle was observed to have a direct bearing on the rapidity with which bronchiectasis developed. If the shadow was small and of fluid-like density and remained so, bronchiectasis developed rapidly (case 11). If the density disappeared and recurred at frequent intervals, dilatations did not develop or were very slight (case 8). However, if the homogeneous density returned and remained unchanged at subsequent examinations, bronchiectasis developed (case 17). If a portion of the triangular density disappeared or if the triangle was small and dense at one examination and then larger and less dense at an early subsequent examination, bronchial dilatations did not occur or were almost negligible in size even after the process had been present for years (case 9). As a rule there was early fluctuation, but in many instances within a few weeks the shadow became of almost fixed size and density and remained so until the bronchi were dilated. These changes can best be interpreted on the basis of obstruction or drainage of the bronchi. If all of the medium-sized bronchi or the main bronchus to a lobe became occluded, the air distally became absorbed, and collapse of the lobe occurred. If one or more bronchi were drained, air entered a lobule or lobules, and that portion of the lobe was reinflated and appeared less dense on the roentgenogram. These changes form the basis on which roentgen interpretation of an individual stage must depend and which enable one to estimate the success of treatment and forecast to some degree the final result.

EFFECT OF DRAINAGE ON THE DEVELOPMENT OF BRONCHIECTASIS

The success of bronchial drainage at a single stage of the process can be measured by the decrease in density and the increase in size of the triangular shadow or its disappearance and the return of the cardiac shadow to a more nearly normal position. When there has been frequent fluctuation in size and density from the onset of the early acute symptoms, slight or no bronchial dilatations have occurred, even when a bronchiectasis was thought clinically to have existed for years. This was illustrated by case 8 (figs. 4 *A* and 4 *B*), in which the course was parallel with that of case 11 for seven years. This patient has had no pulmonary symptoms for five years. In case 8 the patient was thought to have a tuberculous cavity, and postural drainage was carried out by the family for this long period. Most of the patients did not have postural drainage early, and bronchiectasis developed. In M. N. (case 11), in spite of postural drainage for the past few years, the bronchial dilatations have increased in size. These two processes, that is, drainage of a lobe and lack of drainage, are both demonstrated in case 11. The lower lobe of the right lung drained and cleared spontaneously. That of the left showed a fixed density at subsequent examinations. There has been no sign of bronchiectasis on the right since infancy but large sacculations have developed on the left. C. S. (case 9, fig. 5 *A*), in whom the triangle has constantly fluctuated for five years following postural drainage, now shows absence of the triangle on the right side and very slight if any bronchial dilatations on either side.

When drainage into the main bronchi and trachea is profuse, secondary involvement of adjacent lobes is of occasional occurrence as the secretions spill over. The lobe most often involved is the lower lobe on the opposite side. The roentgen appearance is quite often like that of the previously involved lobe and, frequently, at a single examination, one cannot say which was involved first. Occasionally lobules of the upper lobe are filled (figs. 2 *B* and 5 *C*). At times when filling of an additional lobe occurred in this series there were acute symptoms simulating pneumonia, but often only a slight rise in temperature was recorded.

DIFFERENTIAL DIAGNOSIS

The triangular shadow, in which lack of frequent drainage of the bronchi in an early stage brings about bronchiectasis in children, is roentgenologically little different from collapse of the lower lobe brought about by narrowing of the lumen of the bronchus from external pressure. This has been observed in patients in whom a tuberculous abscess (Pott's disease) pressed on a main stem bronchus, in patients with an enlarged heart (case 20, fig. 14 *A*) and as an accompanying finding in aneurysm (in adults). The dense shadow is due to compres-

sion atelectasis or to the backing up of the secretions with subsequent absorption of air in the alveoli.

The triangular shadow, the lateral sharp borders of which faded as the bronchial dilatations increased and freer drainage was afforded, is apparently subjected to a somewhat similar change by intrabronchial plugging by foreign bodies. M. S. (case 10, age 10) had a right main bronchus only partly plugged for ten months by a glass object which had a wire so attached that it kept the bronchus partly patent at frequent intervals. F. C. (case 15, age 3) had a higher degree of collapse follow-

TABLE 1.—*Analysis of the Atelectatic-Bronchiectatic Triangular Density (Fifty Cases)*

	No. of Cases					
"Pneumonia" (collapse)	41	Bronchial obstruction of lower lobe producing collapse	Bronchial drainage	Clears	A. Only partial collapse	A. Complete clearing or only slight tubular dilatation
Anesthesia	1			Recurs	↓ Bronchial drainage stopped	
Foreign body	3			Fails to clear entirely	↓ B. Complete collapse	B. Bronchiectasis, saccular dilatation
Subphrenic infection	1					
Pressure of adjacent structures	2					
Lack of expansion of the lung at birth	2		No bronchial drainage			
Roentgen appearance	Triangular shadow with linear lateral border may reach costophrenic angle; heart and diaphragm retracted toward triangular density; foreign body may be visible		Triangular shadow larger when bronchi are empty, smaller when bronchi are plugged		A. Marked fluctuation of density or B. Small fixed triangular shadow	A. Loss of triangular shadow with scarring of hilus B. Increased size of areas of decreased density within triangle, loss of linear lateral margins of triangle
Clinical diagnosis	Pneumonia, mediastinitis, foreign body, encapsulated pleural fluid, tuberculosis		Bronchitis or infection of upper respiratory tract. bronchiectasis, pulmonary abscess		Bronchitis, infection of upper respiratory tract, mediastinitis, mediastinal empyema, bronchiectasis, tuberculosis	A. Normal chest or B. Bronchiectasis
I	II		III		IV	V

ing complete plugging by a different kind of foreign body similarly placed. This object was present for nine months, and bronchial dilatations occurred. In the former case the triangular shadow was large and of only moderately increased density, and did not have the fuzzy lateral borders found in the late stages of other proved cases of bronchiectasis in which a diagnosis of pneumonia was made at the onset. The other slight variations are probably due to the difference in the obstructing materials. The mechanism of obstruction, as produced by thick fluid-like secretions, could hardly be duplicated perfectly by a formed object.

A slightly similar but rare triangular shadow with a bulging lateral border (hypotenuse) has been observed in tuberculous pneumonia of a lower lobe. While there was compression of portions of the lobe

producing some collapse, most of the density was due to a swelling of the pulmonary tissue and the presence of a central area of necrosis. Because of the increased volume of the abscess, no displacement of the heart toward the area of density occurred until the fluid made communication with the bronchus and escaped. Then the heart was drawn slightly toward the involved side. The large air-filled space on the roentgenogram made the condition evident.

Conditions outside of the lower lobe should not generally be confused with a collapsed lower lobe. When there are solitary collections of fluid in the pleural cavity or mediastinal space, the adjacent structures are displaced away from the triangular shadow, and the recorded density of the shadow on the roentgenogram is directly proportional to its thickness in the course of the penetrating ray. The shadow is dense when large, and proportionately less dense when small. Just the opposite is true in collapse. When the roentgen shadow is large, it is less dense, and it is of greater density when small. The adjacent structures are displaced toward the triangular shadow. This displacement corresponds to the degree of collapse. While a collection of fluid and a chronically collapsed lower lobe might simulate each other when there is a minimum of displacement of the surrounding structures (case 17), as a rule, later roentgenograms will reveal a difference on this basis. In all of the cases observed early, the cardiac displacement has been a constant associated finding. Triangular shadows, due to solitary collections of fluid either in the mediastinum or in the mediastinal pleural space, are rare and when present do not have as smooth linear well defined lateral borders extending from the pulmonary roots to the diaphragm. In two illustrations of proved cases of mediastinal fluid reported by Wessler and Jackes [5] the shadows are elliptic, and there is marked contralateral displacement of adjacent structures.

A collapsed anomalous lobe would be expected to be much smaller and to produce less displacement of adjacent structures but might have the same features as other collapsed regular lobes. While normally anomalous lobes are frequently present in the mesial portion of the base of the lung, those producing dense shadows are unusually rare. At necropsy, the lateral margin of an anomalous lobe has most often presented a convex, almost vertical, lateral border, and its base is narrower than its body. Clinically, however, there should be no difference between an involved anomalous and a similarly involved regular lobe.

A faint triangular shadow secondary to a subphrenic infection has been seen (case 6). During life, in addition to abdominal symptoms, the patient had cough, expectoration and many moist râles similar to the symptoms in the intermediary stages of cases in which visible bronchial dilatations developed later. In other cases a triangular shadow complicating a subphrenic infection has disappeared without evidence of

damage when the pocket beneath the diaphragm was drained. In these cases, the triangular shadow, which represented the collapsed lower lobe, soon became obscured by the upward migration of the exudate from the diaphragmatic pleural space. While the infection seemed to approach the lobe from the pleural space, there was collapse, with an accumulation of fluid in the bronchi. At necropsy its appearance was grossly like that of other patients in whom the onset was associated with a primary pulmonary infection or with an infection of the upper respiratory tract.

OBSERVATIONS AT NECROPSY IN EARLY CASES

An atelectatic lower lobe was found which was usually less than one half the size of the average normally inflated lobe. This lobe was

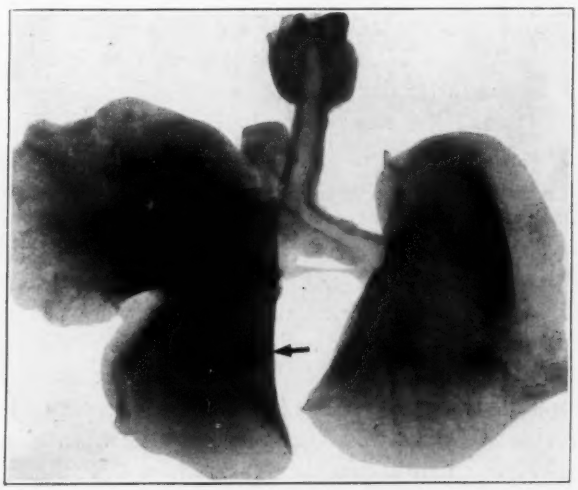

Fig. 15 (case 4, age 2 years, 6 months).—Note the dense triangular outline of the lower lobe of the right lung. There was no evidence of adhesions or of pleural fluid at necropsy.

crowded into the sulcus formed by the lateral side of the spine and the posterior border of the heart. The base of the lobe made contact with the diaphragm which was usually elevated. Figure 15 is a postmortem roentgenogram of the lungs in case 4 in which the homogeneous dense shadow of the right lower lobe was found to be almost airless.

The adjacent lobe or lobes were usually found to pass almost completely around the collapsed lobe on three sides—anteriorly, laterally and posteriorly. Occasionally a slight groove was found on the lateral surface of the collapsed lobe, which had caused the formation of a slight roll or bulge next to the heart. No adhesions or evidence of exudate were found in the pleural space, except in one case (fig. 3 *C*), which occurred secondary to a subphrenic abscess.

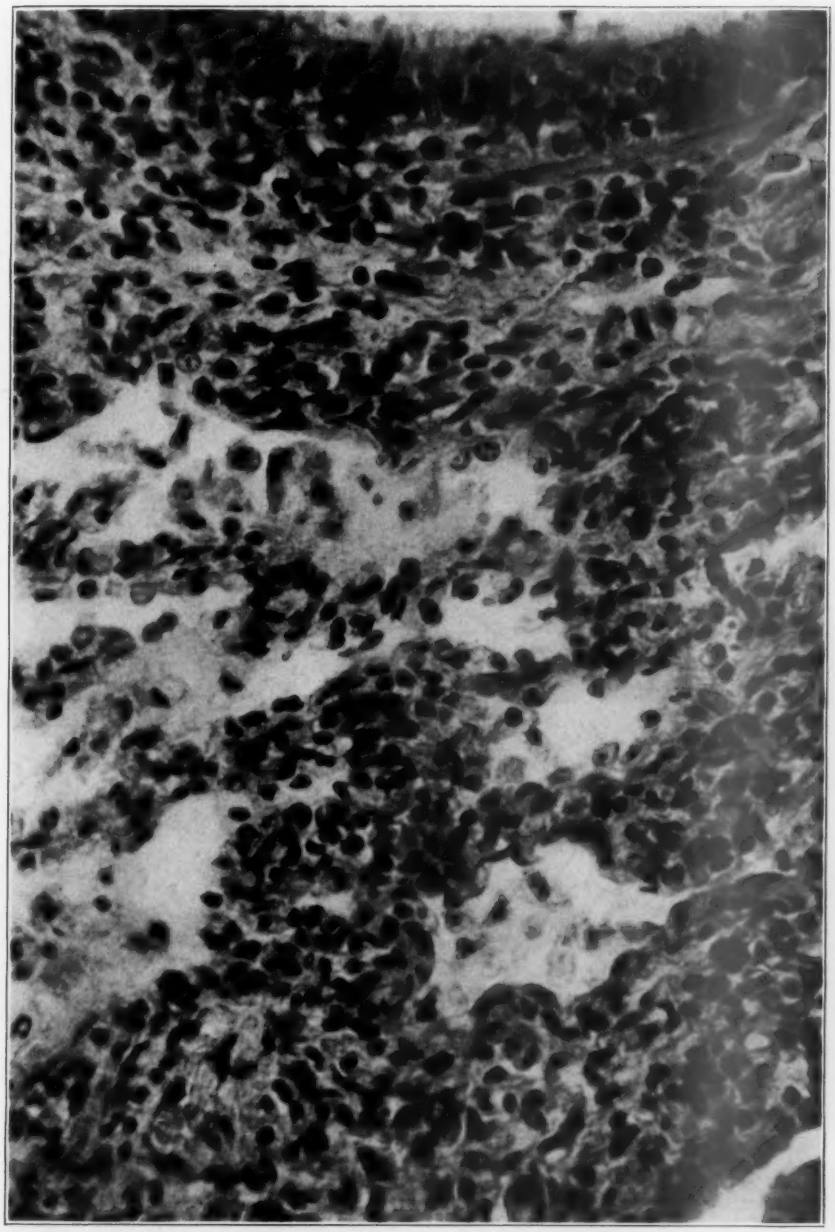

Fig. 16 (case 4, age 2 years and 6 months).—Photomicrograph of tissue from the lower lobe of the right lung, showing minimum changes in the parabronchial and adjacent partial atelectatic tissue; reduced from a magnification of × 500.

The gross changes commonly attributed to well developed cases of bronchiectasis were absent in all the cases which came to necropsy relatively soon after the development of clinical symptoms and of the triangular shadow.

Figure 16 is a photomicrograph of tissue from case 4, showing cylindric epithelial lining with slight changes in the parabronchial tissue

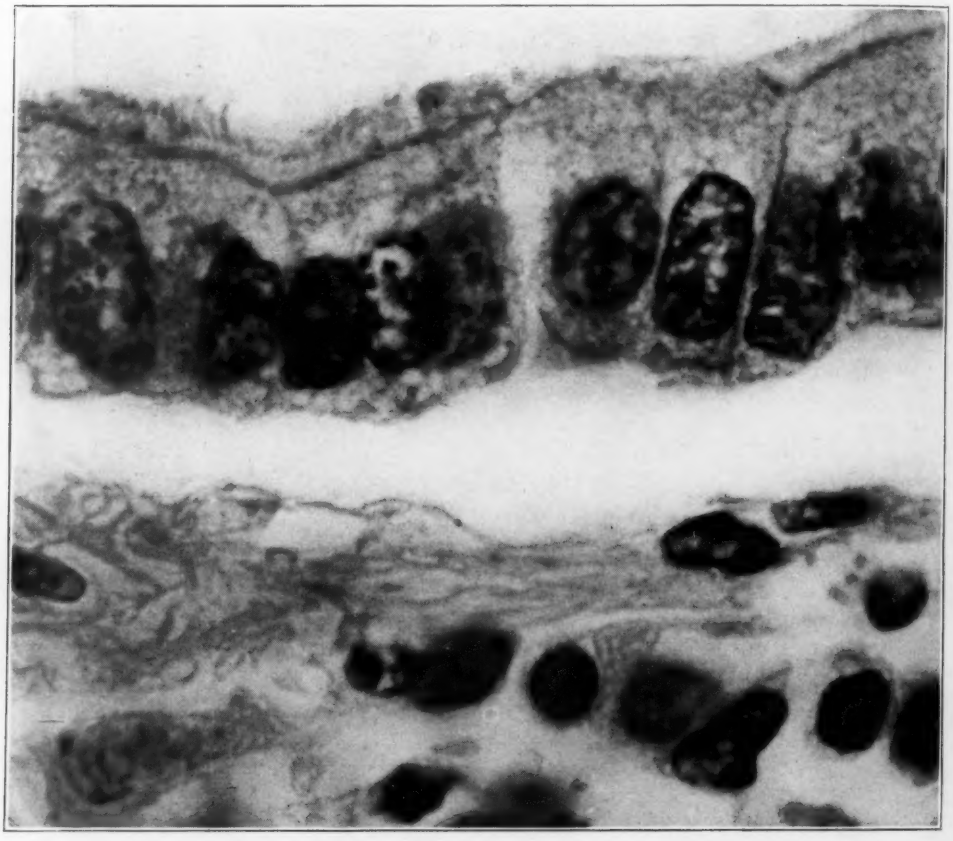

Fig. 17 (case 4, age 2 years and 6 months).—Photomicrograph showing slightly more cubical epithelium; reduced from a magnification of × 2,275.

and slight increase in fibrous tissue which is surrounded by partially atelectatic lung. Figure 17 shows a different area of the same section with cubical epithelium.

Figure 18, a photomicrograph of tissue from case 1, shows tall edematous columnar cells lining the pus-filled bronchus. There is a marked infiltration of polymorphonuclear leukocytes into the adjacent parabronchial and atelectatic pulmonary tissue.

Atelectasis of the lower lobe was present in all the cases that came to necropsy. In each case, a thick gummy exudate, which could be

removed with difficulty, was found filling the lumen of the smaller bronchi. The lumen of the main bronchus was frequently partially patent. Spirochetes, fusiform bacilli and polymorphonuclear leukocytes were found in the exudate.

The pathologic observations recorded at necropsy are summed up as follows: (1) absence of dilatations of the bronchi; (2) absence of

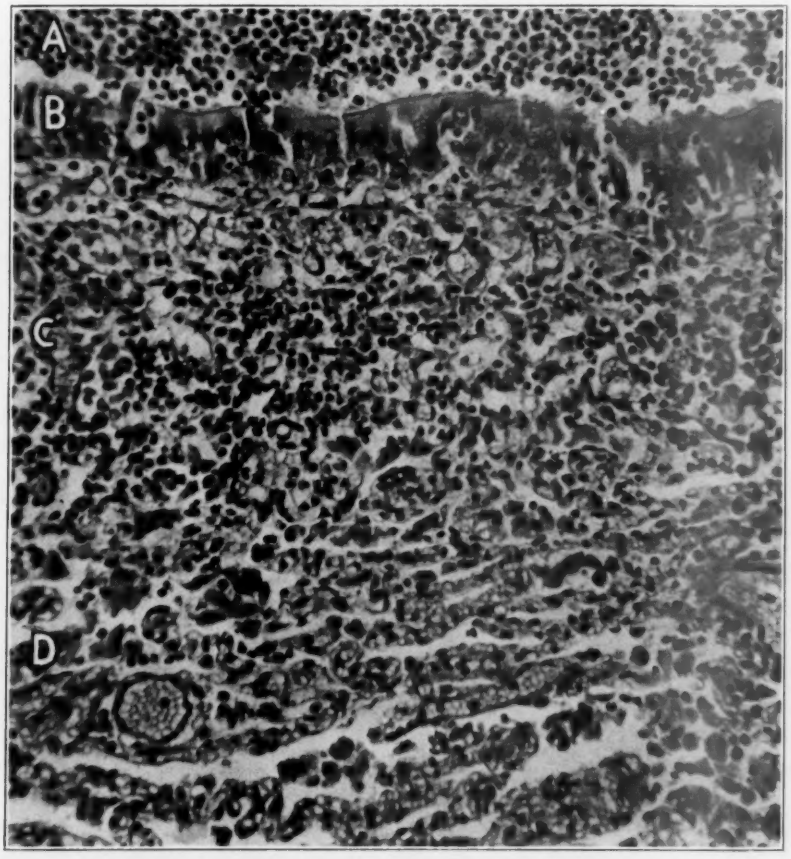

Fig. 18 (case 1, age 3 months).—Photomicrograph showing: *A*, thick exudate in the bronchus; *B*, tall columnar edematous epithelial cells; *C*, marked infiltration of parabronchial tissue by polymorphonuclear leukocytes; *D*, atelectatic pulmonary tissue; reduced from a magnification of × 235.

demonstrable break in, or constriction of, the bronchial wall which maintained its normal contour; (3) absence of distortion of the bronchial wall by enlarged glands; (4) intact condition of the lining, membrane, muscle and cartilage; (5) unusually thick exudate filling the smaller bronchi; (6) slight induration of parabronchial tissue revealed by microscopic examination; (7) frequent small areas of pneumonic consolida-

tion of recent origin in other lobes; (8) atelectasis of the lower lobe, represented by the triangular shadow on roentgenograms, in all the cases. The alveoli of the cut-off section of the lobule had absorbed most of the residual air. Apparently the thick exudate had continued to act as an obstruction and prevented air from entering and reexpanding most of the alveoli.

PATHOGENESIS

With rare exceptions atelectasis accounted for the small triangular area of density in young children in whom the delicate tissues and small caliber of the bronchi formed an ideal situation for an obstruction by tenacious infected secretions. When the air distal to the obstruction of a lower lobe was absorbed by the blood, collapse occurred. This collapsed lower lobe was more inclosed by neighboring structures than normally because the heart was displaced laterally and rotated posteriorly. Not only were the smaller bronchi splintered by thick secretions filling their lumen, but the adjacent bronchi were folded in like the wire ribs of an umbrella, more nearly paralleling and supporting each other as the air was absorbed. In this position, the bronchi were not cleared, and collapse persisted. In the absence of early mechanical drainage, such as that brought about by inverted posture or bronchoscopic aspiration, clearing occurred infrequently. Prolonged crippling of the evacuating mechanism, through infections or pathologic changes due to interference with normal expansion and shortening movements or with the flow of blood and lymph, to allergy, to diminished resistance and resulting pathologic processes or to all these combined, could hardly be without adverse results.

While it is impossible to attribute to each process an exact part in an individual case, it seems that in all probability in these cases bronchiectasis of a lower lobe developed as follows: As the bronchi were filled with infected tenacious secretion, the air present distally was absorbed, and atelectasis resulted. If all, or the greater portion of, the lobules of a lower lobe were collapsed, a more or less triangular shadow appeared on the roentgenogram. If complete collapse was maintained sufficiently long because of delayed drainage or because of a constant supply of thick secretion, pathologic changes occurred which weakened the bronchial walls. The pull of the inspiratory muscles immediately brought about an increase in the negative intrathoracic pressure over the normal because of the decreased intrathoracic volume.[9] This intrapleural pull affected the bronchi of the involved lobe because they, through their "semifluid" filled lumens, were subject to the changes in pressure in the uninvolved portions of the bronchi and trachea and in

9. In all these patients, at necropsy the lower lobe occupied less than half the normal space. In young infants the collapsed lobe was even relatively smaller.

the pleural space. This difference in pressure tended to bulge or stretch the bronchial wall; the weakest portions subject to the greatest amount of damage (often ulceration) ultimately gave way. When the bronchi were dilated, because of their increased volume and the greater plasticity of thin-walled bronchi expiratory pressure at times was more effectively applied, and some evacuation of the fluid into the main air passages occurred. Air then more frequently entered the alveoli of individual lobules, gradually causing partial reexpansion with corresponding nullification of the abnormally increased intrapulmonary negative pressure. This was a rapid or a slow process, probably depending to some extent on the type of infection present, but mainly on the length of time complete collapse existed. It developed more slowly in older subjects. When all these changes occurred, bronchiectasis existed as usually recognized clinically and roentgenologically in older patients.

COMMENT

While I have been unable to find a record of a case in which the signs of atelectasis were followed roentgenologically a sufficient time to show the development of bronchiectasis following an acute pulmonary upset, certain authors have sensed, to some extent, the process brought out by this study. In 1923, Wessler and Jackes,[10] discussing a case of bronchiectasis, said: "It is probable that here the pneumonic process has completely subsided, perhaps years ago, and the disease is now purely a bronchial one." In a recent article, Ballon, Singer and Graham [11] (1932) quoted Hamilton as making the statement, "A fertile source of bronchiectasis in children is pulmonary collapse," and they added, "and it is only now that full cognizance of this remark is being taken."

A satisfactory correlation of the various roentgen findings with the clinical course in these cases has been delayed for two reasons: First, such a correlation would have to cover a period from the onset to the time the usual picture of bronchiectasis has been recognized. This has been hampered by long periods when symptoms were so mild that visits to the physician were likely not to be made, but when roentgenograms were of the greatest importance. Second, a correlation would not be possible without a correct interpretation of the triangular density when seen. The marked decrease in size and the smooth density of the involved lobe apparently have been responsible in a large measure for many mistaken interpretations of mediastinal pleural fluid and mediastinitis. However, one needs only to note the nubbin-like size of

10. Wessler and Jackes,[5] p. 44.

11. Ballon, H.; Singer, J. J., and Graham, Evarts A.: J. Thoracic Surg. **1**:163, 1931.

the lung in the presence of a positive pressure pneumothorax to realize the unusually small size of indivdual collapsed lobes. Yet, in the presence of adjacent hyperexpanded lobes, when the collapsed lower lobe was flattened out against the spine and the posterior cardiac surface, but still made contact with the diaphragm, it has not been generally recognized as a conventional lower lobe.

According to the experience of Coryllos and Birnbaum [12] the size of the involved lobe approaches more nearly that in pure collapse in the presence of organisms of low virulence. It appears that that condition existed in the patients observed, as in many instances there were almost no noticeable symptoms for a period of months during the presence of the unusually small collapsed lobes following the acute symptoms of onset.

The similarity in the general outline of bronchial dilatations within the collapsed lower lobe of children following the injection of iodized poppy-seed oil 40 per cent and the proportionately larger outline of many lower lobes of bronchiectatic lungs in adults following the same type of injections becomes at once apparent. It does not explain all the cases, but it suggests that atelectasis is probably a precursor of adult bronchiectasis in many cases. The early history of acute pulmonary symptoms in so many instances suggests the same sequence of changes as has been followed here in children over long periods.

The scarring of the bronchial walls in cases in which the triangular shadow fluctuated over a period of years, because of frequent drainage and partial reexpansion, and finally cleared without clinical symptoms might be regarded as possibly permanent damage to the lung. A weakened condition or slight dilatation may exist and account for the development of bronchiectasis with associated clinical symptoms in adult life.

The frequent history of pulmonary disease in infancy or early childhood has, no doubt, accounted for the many theories that a congenital bronchiectasis existed. However, it is well to remember that congenital bronchiectasis is rare and has shown no predilection for the lower lobes.[13]

In the past four years I have indexed an unusually large group of cases in which the triangular shadow was present on roentgenograms. All the patients acquiring the shadow in the first year died. All the cases that came to necropsy were considered as showing an acquired atelectasis.

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12. Coryllos, P. N., and Birnbaum, G. L.: Lobar Pneumonia Considered as a Pneumonic Lobar Atelectasis, Arch. Surg. **18**:190 (Jan.) 1929.

13. Koontz, A. R.: Congenital Cysts of the Lung, Bull. Johns Hopkins Hosp. **37**:340, 1925.

The pulmonary tissue had the appearance of tissue that had previously expanded (fig. 16). Although the roentgenologic appearance and the clinical symptoms in infants who succumbed were in every way similar to those of other slightly older children who lived and in whom bronchiectasis developed later, there was a lack of bronchial dilatation at necropsy. Four patients thought to have grossly dilated bronchi of true congenital origin have been encountered during this study. These could be followed almost from birth, and at necropsy little doubt as to their congenital origin existed. Three of these were included in a previous publication.[14] In short, they in no way resembled the cases in this group.

The early and subsequent signs of an atelectatic bronchiectatic process in infants who survive are apparently no different from those occurring in older children, except that the involved lobe appears smaller at first and does not have a growth proportional to that of the adjacent lobes during the early and more rapid period of body growth. The effect is that the involved lobe seems relatively smaller in infancy.

Older children have larger bronchial lumens, less delicate pulmonary tissues and more rigid thoracic walls; they change their position more often and have greater expiratory force. These factors probably account for the more frequent clearing and survival of older children.

In cases in which the triangular shadow, observed early, fluctuated markedly in size and density from one examination to the next, only tubular bronchiectasis developed (case 9), or the chest cleared with little or no evidence of dilatation (case 8). This emphasizes the importance of early persistent attempts at drainage. However, when drainage did not occur with frequent reexpansion of the involved lobe or lobules, saccular bronchiectasis resulted. It appears that in the former instance the bronchi, which were partially patent, at least at intervals, did not weaken and give way readily and consequently did not produce bulbous dilatations. The changes in the walls of the bronchi, in cases in which the triangular shadow fluctuated frequently from the onset of symptoms, seemed to be due, not to localized ulcerations, but to a predominance of fibrosis either in or adjacent to the walls, similar to that occurring in the main bronchial trunks in all the cases of long standing, in which the secretion seemed only to bathe the surface at intervals. Saccular bronchial dilatations occurred in the finer, more distal bronchi, in which thick secretions, possibly aided by mild swelling of the lining membrane, could easily have produced complete obstruction. The presence of stag-

14. Anspach, W. E., and Wolman, I. J.: Large Pulmonary Air Cysts of Infancy, with Special Reference to Pathogenesis and Diagnosis, Surg., Gynec. & Obst. **56**:634 (March) 1933.

nant secretion over a sufficient length of time, either by the germs incubated or by their toxins, would, no doubt, often bring about necrosis or ulceration.[15] This aided in weakening the long immobilized bronchial walls in the more completely collapsed lobe, as found early in cases of saccular bronchiectasis. A condition more amenable to the increased negative intrapleural pull was produced. This resulted when sufficient air distal to the obstruction had been absorbed from the alveoli by the blood to decrease the intrathoracic volume.

Allergy should be considered as possibly assisting in bringing about the initial obstruction to the bronchi by producing a thick secretion or a slightly swollen lining membrane, as observed at necropsy in the earliest cases (2 and 4).

That the size of the dilatations, when once formed, tends to increase in spite of frequent postural drainage was shown by serial injections of iodized poppy-seed oil 40 per cent over a period of years in the individual cases (case 11). The pressure from the weight of retained secretions or increased intrabronchial pressure occurring from the effect of cough [16] is not thought to be sufficient to produce primary dilation.

Bronchiectasis cannot always be explained on the basis of an obstruction to a bronchus by secretions. but the behavior of these triangular shadows, as observed over a long period in a large group, suggested that the obstruction was due primarily to secretion or to secretions secondary to foreign material. In fact, so far as could be determined, primary secretions were the rule in infants and younger children. In every instance at necropsy the smaller bronchi, which grossly appeared quite normal, were filled with thick tenacious secretions.

Small abscesses followed by extensive fibrosis may be present in lobes that are partially collapsed and may later account for bronchial dilatations. However, it is not the rule in cases fitting the clinical and roentgenologic pictures of this group. Small abscesses are less frequently distributed throughout a single lobe. While small areas of collapse may be present about the sporadically placed abscesses, there are, most often during the early course, scattered areas of markedly hyperaerated lung within the same lobe. The small triangular shadow with linear outline has been conspicuously absent. Extensive pyogenic abscesses somewhat simulating the late findings in tuberculosis may permanently alter the alveoli by an extensive fibrosis, and although the involved portion of

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15. McNeil, C.; MacGregor, A. R., and Alexander, W. A.: Studies of Pneumonia in Childhood; Bronchiectasis and Fibrosis, Arch. Dis. Childhood **4**:170, 1929.

16. Mendelsohn's Theory, in Dieulafoy, G.: Manuel de pathologie interne, Paris, Masson & Cie, 1910, vol. 1, p. 98.

the lung is decreased in size, it is not atelectatic. It is not possible for a considerable degree of reexpansion to occur as in the cases illustrated here.

Narrowing of the lumen of the bronchus by enlarged glands, either by preventing drainage or by increasing the temporary intrabronchial expiratory pressure, has recently been regarded as a possible explanation of the development of bronchiectasis.[7f] There were no evidences of the narrowing of the bronchi by glands in any of the cases that came to necropsy.

Many have accepted the belief of Sauerbruch [17] that small dilatations of the middle-sized bronchi exist at birth and that superimposed infection, measles, scarlet fever or whooping cough, makes evident the underlying condition, and a bronchiectasis exists as seen clinically. Necropsy of six patients who died within the first year of life failed to show bronchial dilatation.

I have observed a triangular shadow in an infant six hours after birth (fig 14 *B* and *C*). Serial roentgenograms showed rapid complete clearing, and although the child had passed through a siege of infection of the upper respiratory tract lasting more than a month (patient now 9 months old) no changes occurred in the area of the previously noted density. No other case has been recorded that showed a triangular shadow at such an early period, and no case has been followed over a sufficiently long period to show that this type of case in any way predisposes to the development of bronchiectasis.

Jackson [18] has shown that the material in dilated bronchi resembles the sediment in a septic tank; that is, the lowest layer is of greater consistency than the more superficial layers, which represent the material most recently added. It is the latter material which is most often expectorated. The upper levels of the individual bronchi, therefore, have their lumens evacuated at times. This kind of material was found occluding the fine bronchi at necropsy in fairly early cases in which no dilatations had as yet occurred. Drainage before dilatations had occurred could possibly be obtained by changes in posture, but drainage would be considerably more difficult after the distal portion of the bronchial lumen had enlarged beyond its diameter proximal to the portion filled with exudate. Bronchoscopic aspiration appears to be the only method of completely removing the sediment in advanced cases.

The exact method of producing atelectasis which would be maintained long enough to bring about bronchiectasis, such as occurs in children,

17. Sauerbruch, F.: Bronchiectasis, Etiology and Pathogenesis, Arch. f. klin. Chir. (Kongressbericht) **148**:721, 1927.

18. Jackson, Chevalier: The Bronchiectatic Septic Tank, Chicago M. Soc. Bull. **36**:275 (Nov.) 1933.

could be duplicated only with great difficulty in animals. The dog, which has been used for the experimental production of massive collapse,[19] has, shortly after birth, an evacuating mechanism almost equal to that of an adult, in whom the primary shadow caused by atelectasis persisting over long periods is indeed rare. Infants lie asleep for hours on their backs, have an unusually pliant thoracic wall for months after birth, and are prone to long-standing infections of the upper respiratory tract. These characteristics appear to be an essential requirement of any animal selected for this purpose. Van Allen,[20] Lee[19a] and their co-workers were able only in rare instances to occlude a bronchus in dogs by gummy materials, even under complete anesthesia and complete abeyance of the cough reflex. Although collapse was produced for a short period, the materials were promptly expelled, and reinflation of the lung occurred. To produce the process illustrated in the majority of cases here reported, in which no foreign body was present, it would be necessary to maintain atelectasis, not by obstructing the main bronchus to a high degree, but by occluding individual bronchi in their finer divisions. Otherwise a slightly different picture seen following long-standing obstructions by large foreign bodies (case 15) would be produced. A persistently collapsed lobe becomes an overwhelming burden in infancy because the bronchial lumens are unusually small and, when they are occluded by highly viscous material, sufficient force to clear them is wanting. A better evacuating mechanism probably accounts for the less frequent occurrence of persistent collapse later in childhood and for its extreme rareness in adults.

Particles of food, cotton fibers and short blades of grass have been known to produce collapse which was maintained for an unusually long period and occasionally brought about bronchiectasis in children. The material in itself was not of sufficient bulk to obstruct the bronchi as in collapse produced experimentally in dogs by solidly plugging the lumen of a main bronchus. It appears that the addition of secretions, either previously within the respiratory passages or as a product of local reaction to the foreign material, completed the obstruction sufficiently to bring about persistent collapse and the gradual development of bronchiectasis. If the second or third divisions of the bronchi were reached by tenacious secretions a foreign body would not be necessary because of the small size of the bronchial lumens. The mildly swollen

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19. (*a*) Lee, W. E.; Tucker, G.; Ravdin, I. S., and Pendergrass, E. P.: Experimental Atelectasis, Arch. Surg. **18**:242 (Jan.) 1929. (*b*) Van Allen and Lindskog, G. E.: Obstructive Pulmonary Atelectasis, ibid. **21**:1195 (Dec.) 1930. (*c*) Van Allen, C. M., and Wu, Ching: Increased Elastic Tension of the Lung in Experimental Pneumonia, J. Clin. Investigation **11**:589 (May) 1932.

20. Van Allen and Lindskog.[19b] Van Allen and Wu.[19c]

lining membrane and peribronchial inflammation, as found at necropsy, might have helped in maintaining the collapse by interfering with the normal peristalsis of the bronchi. If the inflammation alone produced the general obliteration of the smaller respiratory passages in a lobe, it would not result in a shadow of markedly decreased size at the onset. The position of the involved lobe would more nearly simulate that usually noted in lobar pneumonia; that is, the lobe would occupy more nearly its normal size and position.

After studying the pictures seen at necropsy in early cases and observing other apparently similar cases over long periods, I have concluded that a mechanical obstruction accounts for the majority, if not all, of the cases of persisting lobar atelectasis producing a triangular shadow at the base of the lungs. I agree with Ehrenberg [21] that atelectasis, especially that which develops in adult chests, cannot always be explained as due to an obstruction of the bronchus by secretion, but that it may be caused by many other factors. It is impossible to estimate the part played by allergy, bronchial spasm and vasomotor disturbances in an individual case. The lack of spontaneous relief for months in some children without areas of reaction producing hilar shadows, as is often seen in so-called "vegetable protein" reactions following the aspiration of certain foreign bodies or chemical substances, supports the theory of a mechanical obstruction in cases in which the patients continued to live but which were comparable in every way to cases that came to necropsy. Unusually thick exudates have frequently been found in cases of tracheobronchitis of nondiphtheritic origin, especially in infants and in small children when it has been necessary to remove this exudate in order to prevent occlusion of the intubation tube.[22] Apparently an exudate of much less viscosity could primarily occlude the fine bronchi, and a constant supply would easily maintain a high degree of obstruction and produce collapse.

SUMMARY

An atelectatic bronchiectatic process of the lower lobe has been studied through correlation of necropsy, roentgenologic and clinical studies throughout life in some cases and from infancy to puberty in others. Conclusions are based on fifty cases, twenty of which are reported and illustrated.

At the onset, there are frequently symptoms and signs simulating a short siege of pneumonia (collapse) followed by slight fever or even

21. Ehrenberg, G. E.: A Critique of Atelectasis in Pulmonary Tuberculosis, Am. Rev. Tuberc. **28**:457, 1933.

22. Gittins, T. R.: Laryngitis and Tracheobronchitis in Children: Special Reference to Non-Diphtheritic Infections, Ann. Otol., Rhin. & Laryng. **41**:422 (June) 1932.

TABLE 2.—*Tabulation of Observations*

Case	Sex	Age When		Living Now	Age at Death	Autopsy			
		First Seen	Last Seen			Performed	Atelectasis	Bronchiectasis	Foreign Body
1	M	3 mos.	4 mos., 1 day	0	4 mos., 1 day	+	+	0	0
2	F	13 days	16 days	0	16 days	+	+	0	0
3	F	6 mos., 2 days	6 mos., 6 days	0	6 mos., 6 days	+	+	0	0
4	M	2 yrs., 6 mos.	2 yrs., 7 mos.	0	2 yrs., 7 mos.	+	+	0	0
5	M	10½ mos.	11½ mos.	0	11½ mos.	+	+	0	0
6	F	2 yrs., 3 mos.	2 yrs., 4 mos.	0	2 yrs., 4 mos.	+	+	0	0
7	F	15 mos.	20 mos.	0	20 mos.	0	—	—	—
8	F	15 mos.	13 yrs., 6 mos.	+	—	—	—	—	—
9	F	2 yrs., 8 mos.	6 yrs., 4 mos.	+	—	—	—	—	—
10	F	9 yrs.	9 yrs., 9 mos.	+	—	—	—	—	—
11	F	3 mos.	13 yrs.	+	—	—	—	—	—
12	F	5 yrs.	8 yrs.	+	—	—	—	—	—
13	F	4 yrs., 6 mos.	7 yrs., 6 mos.	+	—	—	—	—	—
14	M	5 yrs., 6 mos.	8 yrs., 6 mos.	+	—	—	—	—	—
15	M	16 mos.	16 mos., 3 days	0	16 mos., 3 days	+	+	+	+
16	F	6 yrs.	6 yrs., 3 mos.	+	—	—	—	—	—
17	F	7 yrs., 2 mos.	7 yrs., 11 mos.	+	—	—	—	—	—
18	M	4 yrs.	4 yrs., 5 mos.	+	—	—	—	—	—
19	M	10 yrs.	11 yrs.	+	—	—	—	—	—
20	F	10 mos.	18 mos.	+	—	—	—	—	—
21	F	6 hrs.	4 mos.	+	—	—	—	—	—
22	M	4 yrs., 8 mos.	4 yrs., 10 mos.	+	—	—	—	—	—

TABLE 3.—*Tabulation of Observations*

Case	Roentgen Findings					Injection of Opaque Oil		Postural Drainage		
	Right	Left	Both Sides	Persistent	Fluctuating	Age	Bronchiectasis	Performed	Age	Clinical Improvement
1	+	0	0	+	0	0	0	0	—	Died
2	Upper lower	0	0	+	0	0	0	0	—	Died
3	+	0	0	+	0	0	0	0	—	Died
4	+	0	0	+	0	0	0	0	—	Died
5	+	0	0	+	0	0	0	0	—	Died
6	+	0	0	+	0	0	0	0	—	Died
7	+	0	0	0	Slight	0	0	0	—	Died
8	0	0	+	0	+	0	0	+	5 mos. 8 yrs.	Well
9	0	0	+	0	+	3½, 4, 6 years	Slight tubular	+	3-6 years	Marked improvement
10	+	0	0	+	+	0	0	0	—	Well
11	0	0	+ Early	Left	Right	8, 10, 13 years	+	Late	8 yrs.	None
12	0	+	0	+	0	5, 6, 7, 8 years	+	Late	5-8 years	None
13	0	+	0	+	0	7 years	+	Late	7 years	Slight
14	+	0	0	+	0	7 years	+	Late	7 years	None
15	+	0	0	+	0	0	0	0	—	Died
16	+	0	0	+	0	0	0	+	16 years	None
17	0	+	0	Late	Early	—	Late	Late	19 years	None
18	0	+	0	+	0	0	0	+	7 years	None
19	0	+	0	+	0	10 years	+	+	10-11 years	None
20	0	+	0	0	+	—	(?)	0	—	Improvement, heart smaller
21	+	0	0	0	+	0	0	0	—	Well
22	+	0	0	+	0	0	0	+	4 yrs., 9 mos.	Well

subnormal temperature and frequently by a nonproductive cough for weeks. Later, the cough becomes productive, especially noticeable on awakening. Acute exacerbations of fever may be associated with secondary involvement of adjacent tissue. In the absence of drainage of the bronchi, sooner or later dilatations are formed within the triangular density. Ultimately the clinical and roentgenologic picture of bronchiectasis, as noted in older subjects, makes its appearance.

CONCLUSIONS

1. The small well defined triangular shadow with associated displacement of surrounding structures toward it frequently noted at the base of the lung in roentgenograms of children represents a lower lobe in various degrees of collapse. This has been shown by necropsy studies, injections of iodized poppy-seed oil 40 per cent, and roentgen observation over long periods. Fluid in the mediastinum and in the mediastinal portion of the pleural space displaces adjacent structures away from the lobe, and, as a rule, a well defined triangular shadow is not seen in the roentgenogram.

2. While anomalous lobes are frequently seen at necropsy, persistent consolidation or collapse of a supernumerary lobe in the region of the cardiohepatic angle is exceedingly rare. The smallest shadows in the cases here reported were found to represent a regular lower lobe.

3. Necropsies of infants in whom the triangular shadow was present during life did not show dilated bronchi, but in children who continued to live, changes in the triangular shadow appeared later, and bronchiectasis developed.

4. If air entered the collapsed lobe early, the triangular shadow fluctuated in size and was larger and less dense in proportion to the amount of inflation. If postural drainage was instituted early, these fluctuating triangular densities, even though present for years, did not always bring about bronchial dilatations. The triangular shadow is not "pathognomonic of bronchiectasis."

5. If the triangular shadow fails to fluctuate, bronchial dilatations may develop within a few months, but frequently the process covers a period of years.

6. These triangular patterns, when outlined by opaque oils, are frequently seen in adults with bronchiectasis and appear to be acquired rather than congenital.

7. The prognosis can be more accurately determined by observing the behavior of these shadows at successive roentgen examinations.

8. Atelectasis precedes and plays a prominent and most constant rôle in the development of a common form of bronchiectasis of the lower lobe.

9. The most common form of bronchiectasis in children is an acquired process.

10. Early and frequent drainage of the bronchi is essential if the development of bronchiectasis is to be avoided.

1150 North State Street.

THE COMPOSITION OF PRIVATE PEDIATRIC PRACTICE

A METHOD FOR KEEPING ADEQUATE CLINICAL RECORDS

C. ANDERSON ALDRICH, M.D.
WINNETKA, ILL.

The tables which make up the substance of this report were made possible by the method which was used in keeping clinical records of all patients seen in private practice. The technic is as follows: Each day while in the office I dictate into an ediphone all experiences of the preceding day. This record includes the diagnosis and such notes as are thought interesting concerning each private patient seen at home or at the hospital, as well as a list of all patients seen in the office. The details about routine calls on feeding cases and examinations are written on the permanent records at the time of examination. My secretary types the ediphone records in duplicate (chart 1), the original copy being gummed on the reverse side. Strips containing information regarding each patient are cut from this gummed sheet and pasted on the permanent records of the children (chart 2). The carbon copy is a diary, used as the basis for a cross-index which is kept in my study at home. In this cross-index file, each card (chart 3) shows consecutively numbered names of the children who have suffered from the disease listed at its head, together with the complications encountered. In this way it is possible to see at a glance what have been the common complications of each disease and also what diseases have caused the common complications. Only a few moments are required, for instance, to tell what percentage of cases of grip are complicated by otitis media, or, on the other hand, what percentage of cases of otitis media result from grip.

By using this method, which requires only a few minutes daily, I have accumulated during the past few years a considerable mass of information, a superficial summary of which is attempted here. It will be noted that in the number of conditions seen, private practice compares favorably with a hospital service, although there is marked difference in the relative frequency of the various diseases. This is particularly noticeable in the table on the causes of death, in which are found few of the common causes encountered in hospitals. Since these data come from a practice in a fairly well-to-do suburban community, it may be assumed that hygienic and nutritional factors account for some of this difference. This report is submitted as a sample of the relative frequency of the diseases and conditions encountered by the pediatric prac-

CHART 1.—*Daily Ediphone Record*

2-11-33: Buddy Knight—glands are no larger. There is some tenderness back of the left mastoid I thought. White blood cells, 13,000.

2-11-33: Mary Wilson has otitis media with no bulge and a fiery red drum membrane, but no fever.

2-11-33: Philip McNulty has pharyngitis.

2-11-33: Thomas Hough—seen in the office with pain in the abdomen. His liver is swollen and definitely tender when I touch it, and he has had some intestinal upset. The condition is not appendicitis.

2-12-33: Walter Bachrach—a bowel upset and a temperature of 104 F. Is having a diarrhea of an infectious nature.

2-12-33: Vincent Bliss—throat is redder than it was yesterday. There is still no rash.

2-12-33: Anne Powers has recovered.

2-12-33: Allen Gotlieb has the chickenpox.

2-12-33: DeFries Holmes has the grip.

2-12-33: Betty Ruggles had a pain in the abdomen with nausea at noon. All afternoon she had pain in the abdomen in the lower part, no actual vomiting, a temperature of 99 F., definite tenderness in the lower right quadrant, 15,000 white blood cells. Dr. Barrett saw her with me in consultation. He agreed that the condition was acute appendicitis, and took her to Henrotin Hospital. The symptoms were much better following an enema, and I advised that if the white cell count decreased the operation be delayed. On 2-13-33, the count was 12,000. The operation was not done. I have advised that it be delayed indefinitely.

2-13-33: James Woods has an infectious diarrhea.

2-13-33: Tom Carney has a boil on his leg.

2-13-33: Vincent Bliss has a sore throat definitely typical of scarlet fever with no rash on his body.

2-13-33: Nancy Bagley's throat looks much better and the gland is smaller.

2-13-33: Paula Bliss is recovering with no fever now.

2-13-33: Baby Pierson—a new feeding case.

CHART 2.—*History Card Showing Daily Progress*

12-20-32: Robert Cave has definite signs in the lower part of the right side of the chest. I think the condition is going to be lobar pneumonia. I cannot be sure. Temperature ranged from 101 to 105 F.

12-21-32: Robert Cave has a septic temperature with the pneumonia, which is definitely in the lower part of the right side of the chest. Quite toxic and delirious part of the time.

12-22-32: Bobby Cave had a fairly good day. His temperature was quite high, however.

12-23-32: Bobby Cave had a normal temperature in the morning and seemed to have had a crisis, but during the day his temperature went up again to nearly 105 F., and he was irrational all day. His neck is not stiff and his reflexes are active. I think he is having a very toxic case of lobar pneumonia.

12-24-32: Bobby Cave is still very irrational, but his temperature dropped somewhat. It has been only 103 F. at the peak.

12-25-32: Bobby Cave has had his crisis. He is better and rational.

12-26-32: Bobby Cave has a normal temperature.

CHART 3.—*Cross-Index from Scarlet Fever List Showing the Complications*

SCARLET FEVER

29. Mason Warner—measles, cervical adenitis
30. Donald Warner—measles, otitis media, cervical adenitis
31. Richard Johnson—cervical adenitis, otitis media
32. Robert Johnson—cervical adenitis, otitis media
33. Betty Mercer—cervical adenitis
34. Sam Earle—cervical adenitis, measles
35. Bradley Thomas
36. Shirley Hammond—cervical adenitis
37. Lois Blair—cervical adenitis, otitis media
38. Peter Hammond—cervical adenitis
39. Gretchen Rietheimer—cervical adenitis
40. Anne Hearne—cervical adenitis
41. Harry Jackson
42. Jack Ridday

titioner in this particular type of community, as an index to the kind of work for which students are being prepared. The following tables and comments are arranged in the order of their relative quantitative importance in my practice.

The patients in group I, it is estimated, took more time and effort than those in any other group. The general subjects covered in this group are listed in table 1. An attempt was made to arrange each visit so that the mother had sufficient opportunity to explain her problems, feeding or otherwise, and an effort was made not only to answer her

TABLE 1.—*Group I: Routine Care of Infants—451 Patients*

This group includes what are generally called feeding cases. Under this classification are included such items as:

Formulas prescribed, etc.	Pylorospasm
Infantile diarrhea	Colic
Teething difficulties	Habit development
Appetite cultivation	Rickets
Weaning	Hemangioma
Infantile impetigo	Undescended testicles
Tonguetie	Other minor congenital defects, etc.
Infantile eczema	

Data about these can be investigated from the individual histories obtainable from the cross-index

TABLE 2.—*Group II: Acute Infection of the Upper Respiratory Tract—2,165 Cases Plus**

Coryza	232 plus	Conjunctivitis	19 plus
Pharyngitis	643	Retropharyngeal abscess	9
Otitis media	578	Stomatitis	7
Tonsillitis	314	External otitis	3 plus
Cervical adenitis	267	Peritonsillar abscess	2
Laryngitis	252	Glossitis	2
Sinus infections	37 plus		

* In this and other places the "plus" indicates that the condition occurs much more commonly but is not always cross-indexed.

questions, but also to organize the child's program so that preventive advice was given at the proper time, before it was too late.

In group II those cases indexed as coryza presented no other symptoms or physical signs. Many of the other conditions were associated with coryza which was not cross-indexed.

Wherever the throat was generally reddened, the diagnosis of pharyngitis was made, and all types from very mild to extremely severe streptococcic sore throats were included.

All cases of otitis media showed inflammation of the drum membrane and other symptoms. In each instance an abstract of the observations made, the treatment instituted and the results obtained are available for study.

Tonsillitis was diagnosed only when the inflammatory process seemed fairly well localized in the tonsils.

Cervical adenitis was gross in practically every instance, the glands being visibly enlarged. Mere palpable enlargement was not cross-indexed. My observations lead me to believe that there is a type of cervical adenitis which should be classified as an acute infectious disease giving permanent immunity.

Laryngitis includes all cases of ordinary croup.

In all cases designated as sinus infection there was definite evidence of this condition, such as swelling, tenderness and pain. It is realized that many other instances of sinusitis occurred which were not so evident clinically.

Grip, the most common of the infectious diseases to be encountered (table 3), is, to my mind, a cleancut clinical entity. I have not termed it influenza because of my objection to the contraction "flu" and its qualifying adjectives such as stomach, intestinal, etc., which seem to me to lead to diagnostic inaccuracy. The disease here designated grip is distinctly a respiratory disorder of highly infectious nature. It is char-

TABLE 3.—*Group III: Acute Infectious Diseases—1,341 Cases*

Grip	600	German measles	12
Measles	281	Acute anterior poliomyelitis	7
Pertussis	159	Rheumatism	4
Chickenpox	98	Chorea	2
Scarlet fever	65	Erysipelas	2
Roseola infantum	55	Vincent's angina	1
Mumps	55		

acterized by reddened conjunctivae, inflamed nasal membranes, furred tongue and high fever at the onset and reddened pharynx, cough and discharge from the nose later; it is complicated most frequently by otitis media, capillary bronchitis and laryngitis.

Chickenpox is a much more frequent disease than indicated here, because in many cases instructions for treatment were given over the telephone, and a record was not made on the cross-index file.

Roseola infantum shows a surprisingly high incidence. This is partly because I have been intrigued by the possibility of diagnosing it before the exanthem appeared. Whenever an infant was seen without pyuria and when there was no adequate explanation for fever, the mother was requested to report any rash seen within four days, and the possibility of roseola was explained. It was surprising how many times the desired response was obtained and the incorrect lay diagnosis of measles avoided.

No epidemic of German measles occurred during the period covered by my records. In my experience this disease is seen in both sporadic and epidemic forms.

Acute anterior poliomyelitis shows a low incidence because there has not been a real epidemic of this disease in my community during the period of these observations.

Rheumatism and chorea, being class diseases, are met infrequently in this sort of community. Most of these patients were seen in consultation and not in routine practice.

It will be noted that not a single case of diphtheria was seen. This was not the result of adequate immunization, as I have never felt justified in using toxin-antitoxin because of its horse serum content. Only after toxoid became available did I begin immunization for diphtheria as a routine practice. This would seem to be presumptive evidence that the incidence of this disease also is influenced markedly by class or hygienic factors.

Capillary bronchitis, in this report (table 4), is a name applied to a condition which, pathologically speaking, is probably bronchopneumonia, but which presents such mild clinical symptoms that I have not felt justified in using that term, at least not to the parents. Fine subcrepitant râles are heard over more or less localized areas, usually at the base of the lungs, and slight dulness is often elicited. The respiratory rate is but slightly increased, and there is seldom marked cyanosis, dyspnea,

TABLE 4.—*Group IV: Acute Pulmonary Infections—356 Cases*

Bronchitis, upper	180	Pleurisy	12
Capillary bronchitis	127	Empyema	3
Pneumonia	32	Tracheobronchitis, severe	2

pain or bronchial breathing. The temperature subsides after a few days, and the râles become extremely moist, resulting in the "bubbling spring" sort of chest before recovery. This condition complicated two diseases only, grip and measles. It was not followed by pleural effusion or empyema in this series.

Pneumonia includes the lobar and bronchial types and was diagnosed only when frank bronchial breathing was heard in conjunction with the other usual signs.

The pleurisy noted usually, but not always, accompanied lobar pneumonia. It was diagnosed only when definite pleural friction or fluid was demonstrable.

Both cases of tracheobronchitis were of the membranous type, and tracheotomy was necessary. The patients recovered after the usual stormy course.

The conditions listed under group V will not be found adequately stressed in the tables because observations made in routine examinations at the office have not been cross-indexed.

"Cold" vaccines (table 6) were given on the request of mothers to children who were highly susceptible to colds. Good results were not promised, and probably no harm was done.

Rabies vaccine was given only in cases of proved rabies in the offending dog.

Under the classification of vomiting attacks (table 7) were listed cases of active and persistent vomiting usually unassociated with symptoms referable to the upper respiratory tract. These attacks frequently occurred in epidemics, and the condition appears infectious since often an entire family will be affected, not simultaneously, but in sequence. Ketosis usually is present to a more or less marked degree. Certain children seem highly susceptible, and it may be that under this heading a few cases of what others might term cyclic vomiting could be found. I

TABLE 5.—*Group V: Routine Examinations—1,032 Visits* *

Postural defects	Knock knees
Tonsil and adenoid abnormalities	Rachitic deformities
Functional heart murmurs	Nasal infections
Overweight and underweight estimations	Pirquet and Schick tests
Flatfoot	

* Does not include visits of patients in routine care group.

TABLE 6.—*Group VI: Preventive Treatment—813 Cases*

Vaccination for smallpox	395	Measles, parent's whole blood	27
Immunization for diphtheria	300	"Cold" vaccine	26
Pertussis vaccine	61	Prophylaxis for rabies	4

TABLE 7.—*Group VII: Gastro-Intestinal Disorders—474 Cases Plus*

Vomiting attacks	179	Impacted feces	3
Infectious diarrhea	122	Duodenal ulcer	2
Abdominal pain	83	Hemorrhoids	2
Vomiting with clay-colored stools	39	Pyloric stenosis	1
Pylorospasm	21	Food poisoning	1
Catarrhal jaundice	8	Volvulus	1
Carbohydrate intolerance	7		

prefer the simple term of vomiting attacks to names such as "acidosis" and "stomach flu" which have been applied. It may be interesting to those whose experience is confined to hospital work to know that parenteral administration of fluids was found necessary only twice. Practically all the children responded favorably to treatment with carbohydrates and water by mouth.

Cases of obscure abdominal pain, evidently not inflammatory, were fairly common. It is likely that they were caused by excessive peristalsis in most instances.

The cases of vomiting with clay-colored stools might have been called bilious attacks in less enlightened days. Except for the jaundice, they present all of the symptoms and signs of catarrhal jaundice. In some instances it was seen after exposure to Weil's disease.

Carbohydrate intolerance includes cases in which, I think, without treatment the condition would have progressed to typical celiac disease.

It is a freak of circumstance, possibly, that there happened to be two instances of duodenal ulcer during this period. Both were in school children, and were demonstrated roentgenographically as well as clinically.

The extremely low incidence of pyloric stenosis in this series is in harmony with my previous experience that the disease is rare among patients in this sort of community. Pylorospasm, on the contrary, is commonly seen.

Local infections include boils, infected cuts, etc. (table 8).

The high incidence of toxic erythema was one of many surprises to me. This scarlatiniform rash has been seen to follow all of the common infectious diseases as well as ordinary "colds" and sore throats. Its

TABLE 8.—*Group VIII: Cutaneous Lesions—408 Cases Plus*

Local infections	97	Paronychia	7
Toxic erythema	86	Stye	6 plus
Dermatitis	41	Herpes zoster	6
Ringworm	38	Furunculosis	5 plus
Poison ivy	37	Heat rash	3 plus
Impetigo	27 plus	Pompholyx	1
Eczema	24	Ingrowing nail	1
Insect bites	10	Chalazion	1
Pityriasis rosea	9	Acne	1
Scabies	8		

TABLE 9.—*Group IX: Accidents, Etc.—261 Cases Plus*

Cuts and abrasions	124	Burns	11
Miscellaneous injuries	21	Injuries to teeth	2 plus
Dog bites	20	Cerebral concussion	8
Sprains	20	Bruises	5
Hemorrhage	13	Head injuries, severe	5
Swallowed foreign bodies	13	Hematoma	5
Frost-bite	12	Aspirated foreign bodies	2

frequency is one of the clinical facts demonstrable only by some such method as the one here described. Its differentiation from scarlet fever requires considerable clinical judgment.

Infantile impetigo is not included here.

Under the heading of eczema appear only those cases which were more or less generalized.

Under miscellaneous injuries (table 9) are included such mishaps as strains and mashed fingers, perhaps the most common injury seen in this day of the automobile.

The most frequent location for frost-bite in children is under the chin where tightly buttoned coats push a fold of skin into an exposed position. It took me many years to learn the cause of those queer superficial red blotches under the chin.

Similarly, burns are caused along the upper rim of the ears when on warm spring days removal of head coverings allows the sun to blister these parts which have been carefully protected during the winter months. Under this heading are included all types of burns, the incidence of which is rather low.

To get an adequate idea of the frequency of allergic manifestations (table 10), one must realize that undoubtedly many of the illnesses listed under infections of the upper respiratory tract have an allergic background.

It must be remembered that the number of cases of asthma does not mean that there were that many children who had asthma. Many children had repeated attacks.

Allergic rhinitis has been cross-indexed only in the last few months. Its frequency is much greater than would appear from this figure.

TABLE 10.—*Group X: Allergic Manifestations—289 Cases Plus*

Asthma	109	Allergic rhinitis	11 plus
Urticaria	43	Serum sickness	2
Hay fever	34		

TABLE 11.—*Group XI: Surgical Conditions—121 Cases Plus*

Acute appendicitis	50	Intussusception	4
Fractures	26	Meckel's diverticulum: hemorrhage, inflammation	2
Tonsillectomies	?	Antrum drainage	2
Mastoiditis (6 operations)	22	Hydrocele	2
Inguinal hernia	12 plus	Nasal polyp	1
Peritonitis	5		

It is felt that one of the reasons for the low incidence of serum sickness is the fact that toxin-antitoxin was never used as a routine procedure because I could never see the logic of sensitizing the growing generation against horse serum in order to prevent a disease which was a great rarity in my community. Since toxoid has been available, immunization for diphtheria has become a routine practice.

The high incidence of acute appendicitis (table 11) was a complete surprise to me. It is only when one keeps accurate records that an adequate idea of relative incidence can be obtained. Of the total number of cases, 40 were confirmed at operation and 10 were considered so mild that operation could be avoided or deferred. All the patients recovered.

I have not been able to keep satisfactory records of the number of tonsillectomies performed on my patients because, in spite of fairly careful supervision, wayward tonsils go "the way of all flesh" when I am not aware of it. This subject could be investigated to a certain extent

by reference to the routine examination cards to see how often tonsillectomy and adenoidectomy were advised. I am sure the operation has been performed more often than it has been advised by me.

The number of cases of mastoditis, 22, should be compared with the number of cases of otitis media, 578. However, only those in which there were definite physical signs of involvement of the mastoid were so listed. I am fully aware that the mastoid was infected in a much higher percentage of cases, perhaps in all cases of otitis media. Space does not permit discussion of the indications for opening the drum membrane, but paracentesis was seldom done within less than twenty-four hours of the onset, and then only when complete bulging occurred. A bulging drum membrane alone was not considered sufficient indication. It should also be emphasized that only 6 of the 22 patients with evident mastoditis were operated on, and that these 6 were given adequate time for the condition to clear up before the operation was performed. The only child in the group who died had, in addition to mastoiditis, petrositis,

TABLE 12.—*Group XII: Genito-Urinary Conditions—Sixty Cases*

"Pyelitis"	27	Vaginitis	5
Nephritis	18	Paraphimosis	2
Cystitis	8		

which caused meningitis. These data, therefore, may be taken as representing the results obtained under rather conservative management.

Inguinal hernia is inadequately represented, as a few more cases were listed in the charts of the routine feeding group which are not cross-indexed.

All cases of peritonitis were postoperative, and all the patients recovered.

Strangely enough, the only cases of pathologic change in Meckle's diverticula which I have seen in practice occurred during the period of these records. Both cases were diagnosed before operation, and recovery resulted.

Under "pyelitis" (table 12) are included all cases which were characterized by pyuria and fever. The incidence seems to be rather low, but perhaps I was conservative in estimating the number of pus cells in the urine which justifies the diagnosis. Many of the patients had genito-urinary anomalies.

The number of cases of nephritis is unduly large here because of my particular interest in the subject.

The cases designated as cystitis presented frequency of urination and pyuria unassociated with fever.

None of the cases of vaginitis was caused by the gonococcus.

Most of the cases of malnutrition (table 13) could be found by study of the cards under routine examination. This, to my mind, is a difficult diagnosis to make, as the criteria are indefinite and so many different elements enter into the problem.

All cases of pancreatitis were associated with mumps. One was of particular diagnostic interest because the pancreatitis preceded the parotitis, giving rise to qualms about the possibility of an atypical appendicitis.

Unfortunately, my technic does not indicate the extreme frequency of the incidence of psychologic problems (table 14) and the time devoted to their prevention. This subject takes a major part of the time spent

TABLE 13.—*Group XIII: Nutritional and Glandular Disturbances—Forty-Nine Cases Plus*

Overweight, low basal metabolic rates	19	Hypertension, adolescent	2
Fröhlich's syndrome	7 plus	Parotitis, nonspecific	2
Adenitis, not cervical	6	Small stature	1
Malnutrition	4 plus	Ovaritis	1
Pancreatitis	3	Orchitis	1
Diabetes mellitus	2	Mastitis	1

TABLE 14.—*Group XIV: Disorders of the Nervous System and Psychologic Problems—Eighty-Seven Cases Plus*

Psychologic problems	?	Meningitis	4
Anorexia	48	Stammering	2 plus
Enuresis	?	Mirror writing	1
Convulsions	19	Neuritis	1
Encephalitis	6	Erb's palsy	1
Epilepsy	5		

with feeding cases and routine examinations, and its proper handling is, of course, one of the foremost duties of the practicing pediatrician.

Anorexia was listed only when it was the principal complaint. It happens much more frequently, however, and what has been said in the preceding paragraph applies here to a certain extent. However, chronic anorexia is now rarely encountered among the patients cared for from birth. I believe that this is because of careful and persistent preventive measures.

Enuresis, also, has not been adequately cross-indexed.

On the contrary, a record was made of every child who had a convulsive seizure. It seems to me that this extremely low incidence of convulsions must be explained on the assumption that adequate nutrition, using the term in its broadest sense, is of immense prophylactic value in this regard.

Stammering is inadequately cross-indexed.

Flatfoot (table 15) is not adequately indexed and is a common observation in its natural or symptomless type and as an abnormality causing fatigue and discomfort. Bowleg, however, does not appear at all because it was not observed. This deformity has practically disappeared in modern pediatric practice.

Most of the cases of arthritis were of the traumatic type. Of these, several involved the hip.

TABLE 15.—*Group XV: Conditions of the Bones and Joints—Thirty-Five Cases Plus*

Flatfoot	?	Tenosynovitis	2
Arthritis	15	Osteitis fibrosa cystica	1
Bursitis	8	Osteochondritis dissecans	1
Epiphyseal separation	3	Periostitis	1
Osgood-Schlatter's disease	3	Clubfoot	1

TABLE 16.—*Group XVI: Major Congenital Defects—Twenty-Seven Cases*

Congenital heart disease	11	Cleft palate and harelip	3
Atelectasis	4	Hydrocephalus	1
Mongolism	4	Amyotonia congenita	1
Congenital spastic paraplegia	3		

TABLE 17.—*Group XVII: Chronic Conditions—Seventeen Cases Plus*

Rickets	?	Bronchiectasis	1
Functional cardiac murmur	?	Malaria	1
Endocarditis	7	Phlyctenular conjunctivitis	1
Anemia (marked)	4	Tuberculosis	1
Scurvy	1	Hemolytic jaundice	1

TABLE 18.—*Group XVIII: Tumors—Nine Cases*

Cysts	5	Goiter	1
Tumor of the brain	2	Lipoma	1

Only major congenital defects were cross-indexed (table 16). All of the minor abnormalities are noted on the feeding and routine examination charts.

It is evident from table 17 that chronic diseases are distinguished by their infrequency. No cases of syphilis and only a single case of tuberculosis were recognized during the period of these observations.

Signs of rickets, though not cross-indexed, are often encountered. No instance of florid rickets was seen, however.

Functional heart murmurs were heard so often that they constituted almost an average observation, but their actual frequency cannot be given without exhaustive study of the files.

As will be seen, relatively few gross tumors were observed (table 18). One of the tumors of the brain was successfully operated on, but the other one proved fatal.

In the cases listed as unexplained attacks (table 19) there were symptoms such as have been ascribed to disorders of the thymus, the larynx or the endocrine or sympathetic nervous system, according to the point of view of the observer. All were characterized by sudden, more or less alarming, intermittent seizures in young infants. Those that were treated by irradiation over the mediastinum usually improved soon after the exposure. There was 1 death from such an attack during

TABLE 19.—*Group XIX: Miscellaneous Conditions—140 Cases*

Fever, undetermined origin	41	Poisoning, chemical	6
Unexplained attacks	32	Nosebleed	4 plus
Migraine headache	12	Cyanotic attacks	3
Headache	9	Carotinemia	3 plus
Syncope	7	Acidosis	2
Prematurity	6	Deafness	2
Undiagnosed pain	6	Dactylitis	1
Intestinal parasites	6		

TABLE 20.—*Summary Showing the Frequency of the Various Types of Work (Estimated)*

	Per Cent		Per Cent
Routine care of infants	29	Surgical conditions	2
Infections of the upper respiratory tract	23	Accidents, etc.	1
Acute infectious diseases	14	Genito-urinary conditions	1
Acute pulmonary infections	8	Nutritional and glandular disturbances	1
Routine examinations	6	Disorder of the central nervous system and psychologic problems	0.5
Preventive treatment	4	Conditions of the bones and joints	0.25
Gastro-intestinal disorders	4	Major congenital defects	0.25
Cutaneous lesions	3	Chronic conditions	0.1
Allergic manifestations	2	Tumors	0.05

this period of observation. Going back further over the causes of death in my practice, I find that 5 children have died in this manner—more than from any other one cause.

It should be noted that headache is a relatively rare presenting symptom in childhood.

Instances of marked prematurity only were recorded.

Undiagnosed pain does not include pain located in the abdomen.

Only 2 cases of what I thought to be true acidosis occurred.

In table 20 an attempt is made to show the relative amount of time devoted to each of the foregoing groups. It will be noted that about 40 per cent of the time in this pediatric practice is devoted to preventive work.

Calls on routine feeding cases were not included in routine examinations. This group, therefore, was made up of children more than 1 year

of age. It is felt that under ideal conditions care of the older children should occupy a more important position.

In the same way, calls included under gastro-intestinal disorders, cutaneous lesions and chronic conditions were all made in connection with older children.

The figures for psychologic problems are misleading, as only children who were referred primarily for this reason were included. Psychotherapy is a factor in every contact between pediatrician, patient and mother. Therefore, its relative importance from the standpoint of actual time consumed is hard to estimate. It is certainly a major factor.

Since all children who died during this period of observation were examined at autopsy, the causes of death are accurate. Table 21 is perhaps the most interesting of all, because, to my mind, it justifies pediatric effort. The fact that over a period of a few years there were no deaths from such common fatal diseases as the intestinal disorders of infancy,

TABLE 21.—*Group XXI: Deaths—Eleven*

Meningitis (epidemic with hemorrhage; influenza; streptococcic, petrositis)	3	Congenital atelectasis	1
Mongolism	1	Rheumatic carditis	1
Prematurity	1	Unexplained cause	1
Congenital heart disease	1	Tumor of the brain	1
		Intestinal obstruction	1

pneumonia, the ordinary acute infectious diseases, nephritis and tuberculosis demands more than passing notice. In the first place, it must be remembered that the group, as a whole, represents a particularly favored fraction of the population living under optimal hygienic conditions with all economic factors to their advantage. This table indicates that it is possible, under these conditions, to eliminate from the causes of death all but the most hopeless conditions, most of which are not yet preventable. Hundreds of pediatricians in similar communities are having this sort of experience.

It must not be assumed from the foregoing statements that private practice in such a community is a "bed of roses." A great many of these patients were desperately ill and required painstaking care and thought. Responsibility becomes greater rather than less where a child's death is a public calamity and an error in judgment by the physician is freely discussed by the entire community. The physician is constantly "on the spot." In city practice these discussions tend to die out among the tall buildings.

Death in the case of mongolism occurred principally from a congenital heart lesion.

The patient with rheumatic carditis was seen in consultation in a neighboring town. The child was living under poor hygienic and nutritional conditions.

The death from unexplained cause might have been ascribed to an enlarged thymus, which was found at autopsy, if it were not for the fact that the mechanism of such deaths is little understood. Perhaps it will be more challenging and stimulating to investigation of this important subject if one calls such deaths "unexplained" than if one is satisfied with the diagnosis of status lymphaticus.

COMMENT

When this work was begun it was with the idea of developing data for a teaching course in the practice of pediatrics. As time passed, however, so many interesting avenues for thought have been opened up that the technic has become permanent. The physician who uses such a method will find many advantages. There is considerable satisfaction to be derived from keeping such records as the patients are entitled to expect. Because at the bedside the physician is continually confronted with the thought that the ediphone will have to be faced, his diagnostic wits will tend to be sharpened and more decisive. If a mistake is made, it will become evident a few days later when the cross-indexing is done, so that a continuous running check on accuracy is afforded. Many major points in the incidence of disease and its complications will stand out in bold relief, which is stimulating at least to one's curiosity. This method will make the prognosis of the diseases frequently seen much more certain. Since all patients are not treated alike, a therapeutic check will be available. The method is an implement for endless digging into the mine of knowledge underlying every man's private practice. I hope that this outline may be of some value as indicating some of the emphasis which should be placed in pediatric teaching.

723 Elm Street.

Case Reports

AGRANULOCYTIC SYNDROME IN AN INFANT TWENTY-THREE MONTHS OLD, WITH AUTOPSY

ROBERT A. KILDUFFE, M.D.
Director, Laboratories, Atlantic City Hospital
AND
E. HARRISON NICKMAN, M.D.
Visiting Pediatrician, Atlantic City Hospital
ATLANTIC CITY, N. J.

Granulocytopenia or agranulocytosis, the syndrome first described in detail by Schultz in 1922 under the name of agranulocytic angina, was originally regarded as a condition encountered in women of middle age. In the intervening years, however, it has also been reported as occurring in elderly persons, in men, and, infrequently, in children.

While the literature concerning agranulocytopenia has grown rapidly since 1922, reports of the occurrence of this condition in children are relatively uncommon.

Kato and Vorwald,[1] reporting a case in a child aged 5 years, list twelve references to cases reported in childhood, the authenticity of some of which, however, they question because of the symptomatology described.

Among the more recent reports is that of Friedman and Smith[2] in which the patient was a girl aged 2 years, and that of Empey and Proescher.[3]

As it appears that this condition is uncommon, if not rare, in childhood, the following case is deemed worthy of record.

History.—B. A., white, a girl of Norwegian parentage, was born in the Atlantic City Hospital on Feb. 2, 1931. Both parents were syphilitic; the mother's Wassermann test (Kolmer quantitative) was 4 4 4 4 0, and the Kline test, plus 2. The father's Wassermann test on Jan. 20, 1931, was 4 4 4 4 4; the Kline test, plus-minus. Both had had desultory treatment at irregular intervals.

The Wassermann test of the baby's cord at birth was 4 4 4 0 0; the Kline test, plus 2. Delivery was uneventful and normal in all respects. The infant was breast fed for eight months and completely weaned at ten months. There was

From the Laboratories and the Pediatric Division of The Atlantic City Hospital.

1. Kato, K., and Vorwald, A. J.: Agranulocytic Angina in a Child; Pathology and Pathogenesis, Am. J. Dis. Child. **44**:556 (Sept.) 1932.

2. Friedman, E., and Smith, T., Jr.: Agranulocytosis, with Report of a Case in a Child, Colorado Med. **29**:26, 1932.

3. Empey, L. W., and Proescher, F.: Agranulocytosis: Report of Case with Autopsy Observations, Am. J. Dis. Child. **43**:947 (April) 1932.

no history of previous illnesses or of the child's having been vaccinated or immunized against diphtheria. Two months before the onset of the present illness the patient received one intramuscular injection of a bismuth preparation but there was no history of the administration of arsenic or other antisyphilitic drugs.

For several weeks before the child's admission to the hospital the parents noticed that she seemed pale and did not take nourishment well. On Jan. 9, 1933, one week prior to admission, the baby became feverish, seemed weak and without appetite, and on several occasions vomited after feeding. Two days later the left thumb became red and swollen. The illness was attributed entirely to this swelling; home remedies were used, but as no improvement occurred in the general condition, one of us (E. H. N.) was called on January 15.

Examination.—At this time the child was markedly prostrated, had a waxen pallor and seemed dull and torpid, though conscious. The temperature was 104.8 F., and in addition to the marked redness, swelling, and tenderness in the left thumb, a white, patchy membrane was present on both tonsils.

A tentative diagnosis of septic sore throat with possible invasion of the blood stream was made. As a precautionary measure, in view of the tonsillar membrane, 10,000 units of diphtheria antitoxin were administered, and the child was at once admitted to the hospital.

On admission the temperature was 105 F., the pulse rate, 160, the respirations, 26. The skin had a pale and waxen appearance; the respirations were labored. The baby was stuporous, the general appearance being that of an anemic, toxic, and very ill child.

A direct smear from the tonsils on admission showed neither diphtheria bacilli, streptococci nor Vincent's organisms, and only occasional pneumococci. On cultures, staphylococci and Micrococcus tetragenus were grown. The infected thumb was incised by the resident on service and a small quantity of seropurulent fluid evacuated, of which a culture was not taken.

A blood culture on admission gave no growth for four days; then small numbers of staphylococci were found in the broth flask only; these were regarded as probably resulting from a skin contamination.

The examination of the blood revealed a hemoglobin of less than 20 per cent (less than 4 Gm.) by Haden's method; erythrocytes, 760,000 per cubic millimeter; leukocytes, 950 per cubic millimeter; with a differential count as follows: polymorphonuclears, 1 per cent; lymphocytes, 91 per cent; monocytes, 5 per cent. The stained smear showed moderate anisocytosis with macrocytes predominating and 2 nucleated red cells. Two myelocytes and 1 cell regarded as a Türk's irritation form were also seen.

Course.—The condition of the child did not improve, and death occurred on Jan. 16, 1933, barely twenty-four hours after admission.

Autopsy.—Autopsy was performed three hours after death.

The body was that of a well developed and fairly well nourished white female child aged 23 months.

The skin had a waxen, faintly yellow pallor, and the abdomen showed the puncture mark of an intraperitoneal transfusion. There was a paronychial infection of the left thumb which had been incised and drained. There were no other external marks of interest.

Chest: The subcutaneous fat, which was moderate in amount, was a rather bright pale yellow, which contrasted strikingly with the somewhat reddish-brown color of the muscles. The cervical glands were neither enlarged nor palpable. The structures of the neck were removed en masse. Both tonsils showed shallow

crateriform ulcerations with evidence of necrosis. The trachea, esophagus and aorta presented no abnormalities.

The pleural cavities were smooth and glistening and presented no gross changes, nor was there any increase in the pleural fluid. The lungs were free from gross abnormalities other than a few scattered areas of punctate darkening in color in the lower lobes, probably post mortem in origin. There were no gross pulmonary lesions.

The pericardial sac was grossly normal and contained about 10 cc. of clear, serous fluid. The heart presented no valvular or other gross lesions. The muscle was pale and flabby, and fractured with comparative ease.

Abdomen: The abdominal cavity contained a small amount of fluid blood from the transfusion.

The liver was of normal size and consistency but somewhat pale and, on section, contained very little blood. The gallbladder was filled with dark green bile but showed no gross abnormalities. The spleen was dark with a wrinkled capsule, but presented no gross abnormalities of moment.

The gastro-intestinal tract presented no gross changes. The kidneys were of normal appearance, except that they were somewhat smaller than usual.

The following tissues were removed for histologic study: a cervical lymph node, the tonsils, a rib and the liver. Cultures were taken of the heart blood, pericardial fluid, and finger wound. The pathologic diagnosis was: anemia; agranulocytopenia (clinical and laboratory diagnosis); paronychia; ulcerative tonsillitis bilateral; syphilis, congenital (serologic diagnosis).

The postmortem cultures resulted as follows:

From the infected finger, the tonsils, and the pericardial fluid a mixed growth of Staphylococcus pyogenes and a nonhemolytic streptococcus of the gamma type was obtained. From the heart blood the same type of streptococcus was obtained in pure culture.

A histologic examination of the various tissues removed at autopsy was made.

Tonsils: Both tonsils on section presented the same general picture, namely, a subacute inflammatory process with superficial ulceration and necrosis. Stained for bacteria, the sections showed in the superficial layers enormous masses and clumps of diplococci morphologically resembling the pneumococcus, and also short-chained streptococci. The organisms, in diminishing numbers, were also found in the deeper layers of the tissue.

Cervical Lymph Node: Grossly normal in size, appearance, and consistency, the node on section presented no histologic abnormalities. Bacteria were not demonstrated.

Liver: Stains for fat, bacteria, and Spirochaeta pallida were negative. The sections presented no marked abnormalities, although occasional scattered areas of hyperemia and cloudy swelling were found.

Bone Marrow Smears: The marrow was red, and many immature, maturing, and mature erythrocytes were seen in the smears. Nucleated red cells, many being megaloblasts and gigantoblasts, were present in moderate numbers as well as many polychromatic macrocytes.

Granulocytes were practically absent, being encountered only occasionally.

COMMENT

The original conception of agranulocytopenia as a specific pathologic entity has been modified by the numerous studies and reports

which have been made since interest in this condition was aroused by Schultz' report of the six cases he observed. Indeed, this assumption is open to definite question, as the symptom-complex associated with this condition has been reported in conjunction with a variety of intoxications, chemical as well as bacterial.

The early impression that agranulocytopenia was peculiarly associated with the presence of Vincent's organisms (Spirilla and fusiform bacilli) has not been sustained, as the syndrome has not been uniformly encountered in this type of infection and has been seen in its absence. Moreover, as these organisms are practically ubiquitous in the mouth and are peculiarly opportunists, their presence in the ulcerative lesions of the mouth commonly seen in agranulocytopenia cannot be accepted without reserve as conferring on them an etiologic significance.

Bacterial invasion of the blood stream in the presence of agranulocytopenia has also been frequently reported with organisms identified as belonging to the colon group, as well as with Bacillus pyocyaneus and members of the streptococcus group.

Here, also, any etiologic relationship is difficult to demonstrate for several reasons. In the first place, the organisms encountered present no morphologic, cultural or pathogenic differences which would account for any specific depression of the hematopoietic system which is the characteristic and outstanding feature of the disease. It has not been possible in any instance yet reported to produce the disease by animal inoculation with the strains isolated; moreover, while bacteremias with all the organisms in question are by no means uncommon, the concomitant occurrence of agranulocytopenia is neither frequent nor constant.

While Fried and Dameshek [4] have reported the experimental production in rabbits of persistent neutropenia and bone marrow necrosis following the intravenous injection of overwhelming doses of Salmonella suipestifer, this is an organism essentially pathogenic for animals and not yet reported as having been isolated from any human case of agranulocytopenia.

That the symptom-complex described by Schultz may also result from a profound depression of the hematopoietic center following the administration of certain drugs, notably benzene and the arsenic compounds, has been noted by many observers, and this phase of the question has recently been reviewed by McCarthy and Wilson.[5] Such a

4. Fried, B. M., and Dameshek, W.: Experimental Agranulocytosis: Infection of Rabbits with Salmonella Suipestifer by Way of the Blood Stream, Arch. Int. Med. **49**:94 (Jan.) 1932.

5. McCarthy, F. P., and Wilson, R., Jr.: The Blood Dyscrasias Following the Arsphenamines, J. A. M. A. **99**:1557 (Nov. 5) 1932.

case has been encountered in this hospital [6] and this phase—the association of an agranulocytopenic symptom-complex with intoxications of chemical nature—is now so generally recognized and has been so widely discussed as to require no further comment here.

All the accumulated evidence is in favor of a tentative concept that agranulocytopenia is a nonspecific reaction to an infection or intoxication of unusual virulence or intensity which may result from infection with a variety of organisms as well as from intoxication with varied substances of chemical nature.

On the other hand, the fact cannot be ignored that none of the infections or intoxications so far described constantly produces the symptom-complex in question, and that in many instances the bacteremias encountered may well be coincidental, terminal or agonal in origin. It is necessary to assume, therefore, either the presence of a marked lack of resistance in the case of bacterial infections or of a peculiar sensitivity in the case of the chemical intoxications.

One other conception is possible which, if it cannot be proved on the basis of available evidence, cannot be dismissed on the same grounds, namely, the possibility that agranulocytopenia might be due to the action of some unknown and possibly filtrable virus.

In any event, in the light of present knowledge the condition must be regarded as a syndrome produced by a number of causes rather than as a specific pathologic entity due to a specific etiologic agent. It is for this reason that we prefer to record the present case as an agranulocytic syndrome rather than as a case of agranulocytosis.

It presented an unquestionable and profound depression of the myeloid hematopoietic system, especially marked in the bone marrow, with the consequent production of the characteristic blood picture associated with this symptom-complex.

While it is possible that there had been a bacterial blood stream invasion before death, in view of the fact that no septic foci were found in the viscera, and that the antemortem blood cultured showed only a scanty growth of staphylococci in one culture medium, we are inclined to regard the postmortem bacteriologic findings as representing a terminal and agonal invasion and to consider this case as another illustration of a profound septic intoxication associated with an agranulocytopenic blood picture in the presence of, in its final stage, an overwhelming infection.

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6. Barbash, S., and Kilduffe, R. A.: Granulocytic Aplasia of the Bone Marrow of Uncertain Origin Followed by a Leukemoid Blood Picture, J. M. Soc. New Jersey **26**:318, 1929.

ACUTE MASSIVE ATELECTASIS COMPLICATING PNEUMONIA

IN AN INFANT FIVE WEEKS OLD

J. L. BLANTON, M.D.
AND
G. V. MORGAN, M.D.
FAIRMONT, W. VA.

The original report of acute massive atelectasis was made by Pasteur,[1] who reported the condition as a complication of diphtheria. Bradford,[2] following the World War, again stimulated interest in the subject as a complication of thoracic trauma. Since this contribution, numerous articles have appeared in the medical literature dealing with acute massive atelectasis as a complication in various conditions, particularly as a postoperative complication.

Acute massive atelectasis has been reported as a complication in meningitis,[3] tuberculosis,[4] asthma,[5] poliomyelitis,[6] pneumonia [7] and sup-

1. Pasteur, W.: Respiratory Paralysis After Diphtheria as a Cause of Pulmonary Complications, Am. J. M. Sc. **100**:242, 1890.

2. Bradford, J. R.: The Oxford Medicine, New York, Oxford University Press, 1920, vol. 2, p. 127.

3. Kletz, N.: Massive Collapse of the Lung Complicating a Case of Acute Meningitis, Lancet **1**:179 (Jan. 22) 1927.

4. (*a*) Stoloff, E. G.: Acute Massive Atelectasis of the Lung, Complicating a Fresh Tuberculous Primary Complex, Am. J. Dis. Child. **35**:239 (Feb.) 1928. (*b*) Pachard, E. N.: Massive Collapse (Atelectasis) Associated with Pulmonary Tuberculosis and Tumor, Am. Rev. Tuberc. **18**:7 (July) 1928. (*c*) Gutterdam, E. A.: Atelectasis (Massive Collapse) in Pulmonary Tuberculosis, Southwestern Med. **15**:311 (July) 1931. (*d*) Korol, E.: Etiology and Mechanics of Massive Atelectasis, Am. Rev. Tuberc. **24**:276 (Sept.) 1931. (*e*) Sokol, L. T.: Massive Atelectasis Complicating Pulmonary Tuberculosis, Two Case Reports, ibid. **26**:442 (Oct.) 1932. (*f*) Stivelman, B. P.: Atelectasis in Pulmonary Tuberculosis, J. A. M. A. **99**:1666 (Nov. 12) 1932.

5. Peshkin, M. M., and Fineman, A. H.: Acute Massive Atelectasis and Asthma, Am. J. Dis. Child. **42**:590 (Sept.) 1931.

6. Regan, J. C.: Massive Collapse of the Lung in Acute Poliomyelitis, Lancet **2**:1222 (Dec. 13) 1924.

7. (*a*) Gleich, M.: Lobar Pneumonia Complicated by Atelectasis in a Child with Complete Situs Inversus, New York State J. Med. **31**:11 (June 1) 1931. (*b*) Gwyn, N. B.: Massive Collapse of the Lungs, Internat. Clin. **1**:137, 1926.

puration of the lung [8] and in other conditions that cause collapse of the lung by extrabronchial or intrabronchial obstruction, such as foreign bodies in a bronchus,[9] aneurysm of the aorta,[10] intrabronchial tumor,[11] carcinoma of the lung [12] and enlargement of the thymus gland.[13]

The condition is found most frequently in adults. Cases of atelectasis occurring in early life, other than from obstruction by a foreign body, have been reported by Pasteur,[1] Griffith,[14] Gwyn,[7b] Sante,[15] Stoloff,[4a] Abt,[16] Gleich,[7a] Brennemann,[17] Costello,[13] Kletz,[3] Peshkin and Fineman,[5] Regan,[6] Elwyn and Girsdansky [18] and others.

ETIOLOGY

Numerous theories have been advanced as to the etiology of acute massive atelectasis. The most convincing is intrabronchial obstruction.[19]

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8. Elwyn, H.: Acute Abscess of the Lung Complicated by Massive Collapse of the Lung, Boston M. & S. J. **190**:1108 (June 26) 1929. Churchill, E. D., and Holmes, G. W.: Lobar Atelectasis in Chronic Pulmonary Suppuration, Arch. Surg. **14**:1093 (May) 1927.

9. McCrae, T.: The Physical Signs of Foreign Bodies in the Bronchi, Am. J. M. Sc. **159**:313 (March) 1920. Manges, W. F.: Atelectasis as a Roentgen-Ray Sign of Foreign Bodies in the Air Passages, Am. J. Roentgenol. **11**:517 (June) 1924. Jackson, C.: Pulmonary Collapse Due to Foreign Bodies in the Bronchi, Tr. Am. Lung A. **43**:39, 1925.

10. Ylvisaker, R.: Massive Collapse of the Lung Due to Aortic Aneurism, Report of Two Cases, Minnesota Med. **11**:483 (July) 1928. Francis, B. F.: Small Aneurism Pressing on Left Bronchus with Complete Collapse of the Lung, J. Missouri M. A. **26**:134 (March) 1929.

11. Ashbury, H. E.: Recurrent Massive Collapse of the Lung Due to Benign Intra-Bronchial Tumor, Am. J. Roentgenol. **21**:452 (May) 1929.

12. Kernan, J. D., and Cracovaner, A. J.: Carcinoma of the Lung (with Collapse; Bronchoscopy), Arch. Surg. **18**:315 (Jan.) 1929.

13. Costello, J. P.: Thymic Enlargement as Cause of Atelectasis, M. Clin. North America **11**:583 (Sept.) 1927.

14. Griffith, J. P. C.: Massive Atelectasis (Massive Collapse) of the Lung, with Report of Case Occurring in Infant of Three Weeks, M. J. & Rec. **123**:103 (Jan. 20) 1926.

15. Sante, L. R.: Massive Collapse of the Lung, Radiology **8**:1 (Jan.) 1927.

16. Abt, I. A.: Massive Collapse of the Lung; Report of Case in a Boy Aged Ten Years, Am. J. Dis. Child. **30**:347 (Sept.) 1925.

17. Brennemann, J.: Etiology, Diagnosis and Treatment of Massive Collapse of the Lung and Allied Conditions, Wisconsin M. J. **26**:187 (April) 1925.

18. Elwyn, H., and Girsdansky, J.: Postoperative Massive Collapse of the Lung; Report of Case, J. A. M. A. **79**:718 (Aug. 26) 1922.

19. Jackson, C., and Lee, W. E.: Acute Massive Collapse of the Lung; Discussion of Its Mechanism and Its Relation to Foreign Bodies in Bronchi and Post-Operative Complications, Ann. Surg. **82**:364 (Sept.) 1925. Hirschboeck, E. J.: Massive Collapse of the Lung, with Report of Necropsy, Minnesota Med. **11**:135 (March) 1928. Tucker, G.: Peroral Endoscopy, Surg., Gynec. & Obst. **42**:743 (June) 1926. Coryllos, P. N., and Birnbaum, C. L.: Obstructive Massive Atelectasis of the Lung, Arch. Surg. **16**:501 (Feb.) 1928.

Brennemann [17] expressed the belief that this explanation can account for the vast majority of the cases. Pasteur [1] thought that paralysis of the diaphragm was the etiologic factor in cases of atelectasis following diphtheria. This has been questioned by Jackson and others.[19]

Bradford [2] suggested that there may be a temporary paralysis of the inspiratory mechanism of reflex origin. Scrimger [20] stated that vagal stimulation might be a factor. Briscoe [21] pointed out the influence of posture. Korol [4d] called attention to the fact that prolonged dorsal recumbency may be a factor and stated that acute massive atelectasis can occur without the occlusion of a bronchus. Jones [22] believed that spasmodic constriction of the bronchioles plays an important part in producing the atelectatic condition; that the bronchial plugging may be a result of the massive atelectasis rather than the cause of it. Scott [23] expressed the thought that this bronchial spasm may result from reflex irritation in other parts of the body. However, bronchial spasm as seen in asthma usually produces emphysema instead of collapse. Scott also suggested the possibility of a vasomotor disturbance in the pulmonary vessels with an outpouring of secretions causing obstruction in the bronchioles.

Band and Hall [24] stressed the abolition of the cough reflex and the limitation of respiratory movements as important etiologic agents, acting along with a viscid intrabronchial secretion. Pulmonary embolism may be a factor in some cases as suggested by Gwyn.[7b] Extrabronchial pressure has been an important factor in certain cases. Papillomas of the larynx were the cause of collapse of the lung reported by McIllraith.[25] This diagnosis was confirmed by autopsy.

Various factors may be of importance in certain individual cases; nevertheless, bronchial obstruction stands out prominently as the basic cause of the collapse. Henderson [26] summarized the present situation by stating "the only clearly demonstrated causes are shallow breathing

20. Scrimger, F. A. C.: Postoperative Massive Collapse of the Lung, Surg., Gynec. & Obst. **32**:486 (June) 1921.

21. Briscoe, J. C.: The Mechanism of Post-Operative Massive Collapse of the Lung, Quart. J. Med. **13**:293, 1920.

22. Jones, L. R.: Massive Collapse of the Lung, M. Bull. Vet. Admin. **8**:20 (March) 1932.

23. Scott, W. J. M.: Postoperative Massive Collapse of the Lung, Arch. Surg. **10**:73 (Jan.) 1925.

24. Band, D., and Hall, I. S.: Post-Operative Massive Collapse of the Lung; Clinical and Experimental Study, Brit. J. Surg. **19**:387 (Jan.) 1932.

25. McIllraith, C. H.: Pulmonary Collapse, Consequent on Papillomata of the Larynx; Unrelieved by Tracheotomy; Death, J. Laryng., Rhin. & Otol. **33**:257 (Sept.) 1918.

26. Henderson, Y., et al.: The Treatment of Pneumonia by Inhalation of Carbon Dioxide: I. The Relief of Atelectasis, Arch. Int. Med. **45**:72 (Jan.) 1930.

and the accumulation of mucus, resulting in a mechanical blocking of the air tubes." Undoubtedly this was true in the case that follows.

ATELECTASIS IN PNEUMONIA

Acute massive atelectasis following pneumonia appears to be a rare complication. Bradford [2] stated that it does occur as a complication of this disease. Brennemann [17] and Griffith [14] also spoke of the occurrence of acute massive collapse following pneumonia, but no cases are reported.

Gleich [7a] reported a case in a child, aged 7 years, with a complete situs inversus.

Gwyn [7b] cited three cases, one in a boy aged 6 years. He called attention to the contributory influence of the lagging and disinclination of the respiratory muscles of the affected side to do their work as a possible factor in producing the collapse.

Abt [16] reported a case of postoperative massive collapse complicated by pneumonia in a boy aged 10 years. The pneumonia, however, followed the collapse.

Thoenes [27] reported the occurrence of cardiac displacement toward the involved side during pneumonia in infancy. He stated the belief that atelectasis is a factor in producing cardiac displacement, being aided by the overdistention of the unaffected lung. Wallgren [28] added reports of nine cases occurring during the first two years of life in which cardiac displacement toward the affected side was found during the early stage of pneumonia. He considered the displacement as being due to a diminished volume of the lung and thought that possibly emphysema on the unaffected side was a factor.

Tallerman and Jupe [29] substantiated the foregoing findings during the early stage of pneumonia in childhood. They cited five cases and expressed the belief that the displacement results from traction exercised by shrinkage due to partial collapse of the involved lung aided by the push of the sound lung, which is frequently distended by compensatory emphysema.

The occurrence of this phenomenon during the acute stage of pneumonia and the accompanying lagging and poor expansion of the involved side seem to favor the development of acute massive atelectasis much more frequently than the cases reported indicate.

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27. Thoenes: Mediastinal Displacement in Infiltrative Disease of the Lung in Infancy, Monatschr. f. Kinderh. **22**:353, 1922.

28. Wallgren, J.: Ueber Lageveränderungen des Herzens bei akuter Pneumonie, Acta pædiat. **3**:81, 1923.

29. Tallerman, K. H., and Jupe, M. H.: Displacement of Heart in Pneumonia in Childhood, Arch. Dis. Childhood **4**:155 (Aug.) 1929.

The case that follows is of particular interest in that it occurred in an infant, aged 5 weeks, after the pneumonic process had apparently begun to resolve. This would indicate that the bronchial secretions played an important part in the etiology.

REPORT OF A CASE

History.—P. B., a female infant, was delivered by one of us (Dr. Morgan) following a normal pregnancy and a short easy labor. The patient was the fourth child of healthy parents. She cried lustily at birth, and there was no cyanosis. Physical examination at birth revealed a normal baby, with the exception of a bilateral talipes valgus. The infant was breast fed and thrived well for three weeks. She then contracted influenza, which the other three children had, and which was prevalent locally at the time. Two days later, she had rapid grunting respirations and a temperature of 103 F. Pneumonia involving the lower lobe of the right lung was diagnosed. Four days later the temperature was normal, and the infant apparently was going through the process of normal resolution of the pneumonic area. Three days later, one of us (Dr. Morgan) was urgently called by the parents who stated that the baby was dying. She recovered quickly, and examination of the chest showed an absence of breath sounds and dulness over the region of the right lung. The cardiac impulse was displaced to the right of the sternum. The parents stated that the infant had been having severe coughing spells since she had pneumonia. These spells persisted until she was brought to the Fairmont Hospital No. 3 at the age of 6 weeks, on Jan. 1, 1933.

Examination.—The infant appeared to be perfectly comfortable until she was disturbed; then a paroxysm of coughing not unlike a paroxysm of pertussis occurred. She became quite cyanotic during the attack, and the coughing continued until emesis was produced by gagging. The emesis yielded a thick, tenacious, sticky mucus. The temperature was 101 F.; the pulse rate, 160; respiratory rate, 40. The infant was 52 cm. long and weighed 2,980 Gm. She was well developed but undernourished. There was no dehydration. The head was normal. The anterior fontanel measured 3 by 4 cm. and was level. The posterior fontanel was closed. The ears were normal. There was a serous nasal discharge with mild obstruction. The mouth was normal. The throat was slightly reddened, and there was an excess of mucus in the pharynx. The tonsils were normal. There were no enlarged glands in the neck. The right side of the chest was retracted and almost immobile. The left side of the chest appeared prominent. The cardiac impulse could be seen at the right nipple in the fourth interspace, 3.5 cm. from the midsternal line. Percussion revealed a sound varying from dulness to flatness over the entire right lung. The left side was hyperresonant. Bronchial breathing was heard over the entire lung. There were showers of fine crackling râles when coughing occurred. Fremitus was increased. The heart sounds were clear and distinct. There were no murmurs. On the left there were exaggerated vesicular breath sounds, and there were no râles. The abdomen was soft. The lower edge of the liver was felt 4 cm. below the costal margin, in the midclavicular line. The spleen was palpable. The genitals were normal. On examination the spine was normal. There was bilateral talipes valgus. All the reflexes were normal.

Subsequent Course.—The parents refused hospitalization for the infant because of the difficulty of maintaining the breast feedings. The patient was permitted to return home, instructions being given to the parents to change her position at frequent intervals, to roll her on the unaffected side and to produce crying, and

to give the infant potassium iodide, 0.065 Gm., every four hours. The aim was to render the sputum less viscid.

On January 8 the temperature was 99 F.; the pulse rate, 140; the respiratory rate, 40. The infant weighed 3,192 Gm. She had less difficulty in raising sputum. Examination of the chest showed little improvement. The roentgen examination showed patches of areated lung near the periphery on the right side. The treatment was continued. Cod liver oil and orange juice were added to the diet.

Three days later the temperature was 99 F.; the weight, 3,248 Gm. Paroxysms of coughing and cyanosis had been less frequent. The cardiac impulse was 2 cm.

Fig. 1.—Postero-anterior view, one week after onset, showing increased density throughout the right lung and displacement of the heart and trachea to the right.

to the right of the sternum. There was bronchovesicular breathing over the lower lobe of the right lung, with persistence of bronchial breathing over the upper and middle lobes. Numerous fine râles were heard throughout the right lung.

On January 16, the temperature was 99 F.; the weight, 3,374 Gm. Definite improvement was noted. The cardiac impulse was just to the right of the sternum. There were numerous bubbling râles throughout the right lung in front. Posteriorly, there was persistence of bronchial breathing as low as the tip of the scapula. Fremitus was still increased over this area. Roentgen examination showed further clearing from the periphery toward the hilus. Because of the numerous moist râles at this time, the potassium iodide was discontinued.

Three days later at the request of the parents, who stated that the infant was having greater difficulty in raising the tenacious sputum, medication was resumed.

On February 2, the temperature remained 99 F.; the height was 54 cm.; the weight 3,860 Gm.; the infant still had a productive cough. The cardiac impulse was in the fourth interspace, 3 cm. to the left of the sternum. There was vesicular breathing throughout the right lung, with fine râles persisting over the upper lobe posteriorly.

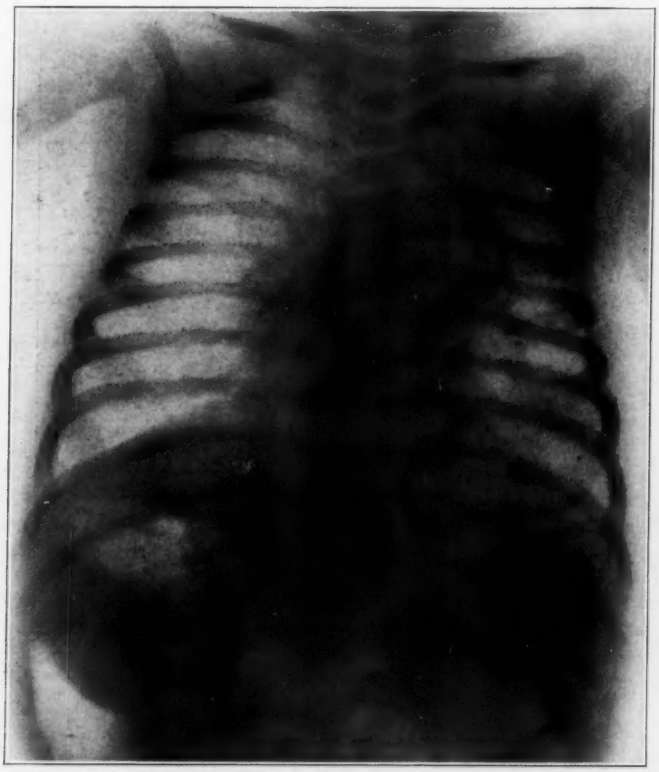


Fig. 2.—Postero-anterior view, three weeks after onset, showing clearing from periphery toward the hilus.

On February 16, the temperature was still 99 F.; the height was 60 cm.; the weight, 4,527 Gm. There was no cough. The infant appeared to be perfectly well. Physical examination gave normal results.

SIGNS AND SYMPTOMS

Sudden onset with cyanosis, rapid breathing, rapid pulse and moderate temperature are the usual symptoms of acute atelectasis. The case herein reported presents a striking picture that has not been stressed. The coughing occurred in paroxysms similar to the paroxysm in pertussis in infancy. There were repeated coughing, marked cyanosis

and relief by emesis of the thick tenacious sputum. The infant appeared relatively comfortable between these attacks, which numbered three or four every day during the first two weeks.

Physical examination reveals a characteristic picture. There is immobility and retraction of the involved side with dulness to flatness on percussion. The breath sounds are either absent or greatly increased and bronchial. Fremitus is increased. The important finding on physical examination is that the heart is displaced toward the involved side.

Fig. 3.—Postero-anterior view, six weeks after onset, showing satisfactory clearing.

The unaffected side is hyperresonant and presents exaggerated vesicular breathing with some increase in fremitus.

As improvement takes place, variation in the auscultatory findings occur. Fine crackling râles appear. Later, coarse and musical râles are heard. There is a gradual transition back to the normal, with the heart returning to its normal position.

The roentgen findings are characteristic of the condition. There is increased density over the involved lung. The heart and trachea are

displaced toward the involved side. The interspaces are narrow and the ribs form more of an acute angle than is normally found. The diaphragm is immobile and elevated on the affected side.

The physical signs together with the roentgen findings differentiate the condition from other pulmonary lesions.

TREATMENT

Sante [30] has suggested that the patient be turned on the uninvolved side and that coughing be encouraged while in this position. He has witnessed prompt clearing up of the condition following this procedure. This plan was followed in the case reported herein so far as practically possible in an infant. In addition, potassium iodide was given to render the bronchial secretion less viscid. That this aided in recovery is confirmed by the parents. The potassium iodide was discontinued at one point during the treatment because of the presence of numerous moist râles over the affected lung. Three days later, the parents requested that the medication be used again. The infant was having greater difficulty in raising the tenacious sputum than while the potassium iodide was being given. A few days later the secretion was less viscid, and the patient felt more comfortable.

Bronchoscopy offers the most satisfactory means of treating patients with atelectasis. It has been stated that on the average recovery takes place in three days following this method of treatment as compared to three weeks with a conservative type of care.[31]

COMMENT

Acute massive atelectasis occurring in infancy is not to be confused with congenital atelectasis, although this type may be massive in character.[32] Congenital atelectasis applies to lung tissue that has never expanded. It is usually bilateral and is found chiefly in the central and posterior portions of the lung. It is present from birth. Dunham [33] and others have shown that large areas of lung tissue may be involved.

Persistence of the cone-shaped thorax of the fetus is one of the characteristic features in the congenital form of atelectasis. Displace-

30. Sante, L. R.: Massive (Atelectasis) Collapse of the Lung, with Reference to Treatment, J. A. M. A. **88**:1539 (May 14) 1927.

31. Hirschboeck, E. J.: Massive Collapse of the Lung with Report of Necropsy, Minnesota Med. **11**:135 (March) 1928.

32. Chapin, H. D., and Royster, L. T.: Diseases of Infants and Children, New York, William Wood & Company, 1929, p. 395. Booth, G. H. H.: A Case of Congenital Atelectasis, Brit. M. J. **2**:56 (July 9) 1932.

33. Dunham, Ethel C.: Atelectasis in the Newborn, Am. J. Dis. Child. **43**:594 (March) 1932.

ment of the heart and mediastinum does not seem to occur.[34] Crying aids in overcoming the cyanosis in the congenital cases, while in acute massive atelectatic cases the cyanosis is increased by any exertion. Crying, however, aids in reexpanding the collapsed lung.

SUMMARY

A case of acute massive atelectasis following pneumonia in an infant 5 weeks old is reported.

The factors favorable for the development of acute massive atelectasis following pneumonia are pointed out.

The differential features between acute massive atelectasis and congenital atelectasis are given.

34. Wasson, W. W.: A Roentgenographic Study of the Infant Chest as Seen at Birth, J. A. M. A. **83**:1240 (Oct. 18) 1924.

CONGENITAL ABSENCE OF THE GALLBLADDER AND THE EXTRAHEPATIC BILE DUCTS

J. D. KIRSHBAUM, M.D.
CHICAGO

Congenital absence of the gallbladder, including the extrahepatic bile ducts, is probably the second in order of frequency of rare anomalies. The presence of two livers is given as the rarest. The case reported here, which occurred in a child 5 months of age, represents 1 in a series of 3,669 postmortem examinations performed at the pathologic institute of the Cook County Hospital since 1928. Because of its rarity, it was deemed worthy of reporting. There are over 175 reports of congenital obliteration or atresia of one or more of the bile ducts and gallbladder, as reviewed by Parsons and Hickmans,[1] Rolleston and Hayne,[2] Thompson,[3] Wyard,[4] Holmes,[5] Ladd,[6] Emmanuel[7] and Coburg.[8] In the cases represented by these reports the gallbladder was usually normally present, or at least a remnant of it. In some cases, one or more of the ducts were recognized. In reviewing the literature on complete absence of the gallbladder, including the extrahepatic bile ducts, there was found a report by Bower,[9] who cited 14 cases and added his own observation. In only 6 of the 14 cases, were the records complete. In the remaining 8 cases, the authors failed to mention whether all the extrahepatic ducts were absent. Three additional cases with postmortem records have been reviewed (Hill,[10] Buschnell[11] and Hansen).[12]

From the Department of Pathology, Cook County Hospital, Dr. R. H. Jaffé, Director.

1. Parsons, L. G., and Hickmans, Evelyn M.: Biliary Cirrhosis, Am. J. Dis. Child. **31**:461 (April) 1926.
2. Rolleston, H. D., and Hayne, L. B.: Brit. M. J. **1**:758, 1901.
3. Thompson, J.: Edinburgh M. J. **37**:606, 1901.
4. Wyard, S.: Lancet **2**:495, 1914.
5. Holmes, J. B.: Pyloric Stenosis in Older Children, with Report of a Case of Pyloric Spasm Ending Fatally, Am. J. Dis. Child. **2**:405 (Dec.) 1916.
6. Ladd, W. E.: Congenital Atresia and Stenosis of the Bile Ducts, J. A. M. A. **91**:1082 (Oct. 13) 1928.
7. Emmanuel: Brit. M. J. **2**:385, 1907.
8. Coburg, H.: Frankfurt. Ztschr. f. Path. **40**:281, 1930.
9. Bower, J. O.: Ann. Surg. **88**:80, 1928.
10. Hill, N. H.: Lancet **2**:654, 1927.
11. Buschnell, F. G.: Lancet **2**:1621, 1905.
12. Hansen, in Jordan, H. E., and Kindred, J. E.: A Text-Book of Embryology, ed. 1, New York, D. Appleton & Company, 1926, p. 231.

Hill's [10] patient, 4 months of age, revealed an absence of the gallbladder and the extrahepatic bile ducts. Buschnell's [11] patient, 8 months old, had been jaundiced since birth. Examination after death failed to reveal a gallbladder, its normal site being replaced by fibrous tissue. In addition, the extrahepatic ducts were missing. A congenital hepatic cirrhosis with an obliterating cholangeitis due to a primary developmental malformation was also present. Hansen's [12] patient was a premature child, 6 weeks of age, who had been jaundiced since birth. Grossly, examination revealed absence of the gallbladder, absence of the cystic duct, fibrosis of the common duct and biliary cirrhosis. With the microscopic evidence of lymphocytes and round cell infiltration, the author then considered that the cause might have been an infectious process producing the obliteration.

REPORT OF CASE

History.—F. W., a Negro boy, aged 4 months, was admitted to the pediatric service of the Cook County Hospital on Feb. 13, 1932. The mother stated that the child became jaundiced one week after birth, the icterus being constant and of the same degree. Several weeks after birth the child bled from the umbilicus. A cold had been present for several weeks but was now clearing up. The stools had been light yellow to green, and the appetite was good. The child was gaining in weight and appeared well save for the icterus. According to the past history the child had had whooping cough. He was a full-term, first-born baby, and had appeared normal at birth. The labor of the mother lasted twenty-four hours. The child was breast fed, supplemented with a bottle (half milk and half water), and was taking cod liver oil. He was of normal development. The mother had had no miscarriages, and the father was well and strong.

Physical Examination.—A fairly well nourished, markedly icteric Negro infant was seen. The temperature was 99.4 F.; the pulse rate, 146; respirations, 32, and the weight, 10 pounds, 2½ ounces (4,592 Gm.). There was a mucopurulent discharge from the nose, and the child was breathing heavily. The anterior fontanel was open. The throat was reddened, and the sclerae were icteric. The pupils were equal and reacted promptly to light. There was no rigidity of the neck, with moderate cervical adenopathy and enlarged axillary glands. The breath sounds were normal, and there was no dulness or râles in the lungs. The heart appeared normal. The liver was barely palpable, and the spleen was questionably enlarged; there was no tenderness or rigidity; no masses were felt, and no ascites were present. The inguinal glands were shotty. The extremities and the reflexes were normal. A diagnosis of syphilis was made.

Laboratory Findings.—Urinalysis showed no albumin or sugar; bile was present (4+). Cultures from the nose and throat were negative for diphtheria. The Pirquet test was negative. There were 13.07 mg. of urea and 106 mg. of sugar per hundred cubic centimeters of blood. The Wassermann reaction of the blood was negative. There was an immediate indirect van den Bergh reaction, which was interpreted as compatible with hemolytic jaundice. There was an increase in the fragility of the red blood corpuscles, which hemolyzed at 0.48. The stools were acholic and a chemical test revealed blood. The white blood count was 11,000; the hemoglobin, 50 per cent; lymphocytes, 74 per cent; polymorphonuclear leukocytes, 25 per cent,

and basophils, 1 per cent. Spinal puncture gave negative results. During the first week the child lost 1 pound (453.6 Gm.), although he was eating well. A roentgen examination revealed a destructive process in the epiphyses of both knees and ankle joints, diagnostic of syphilis; the process later proved to be severe rickets. The child then commenced to have a fever, which reached 104.8 F., with vomiting and diarrhea, and the abdomen became distended. He was restless, cried a great deal, became dyspneic, and died four weeks after admission at the age of 5 months. The final diagnosis was congenital syphilis.

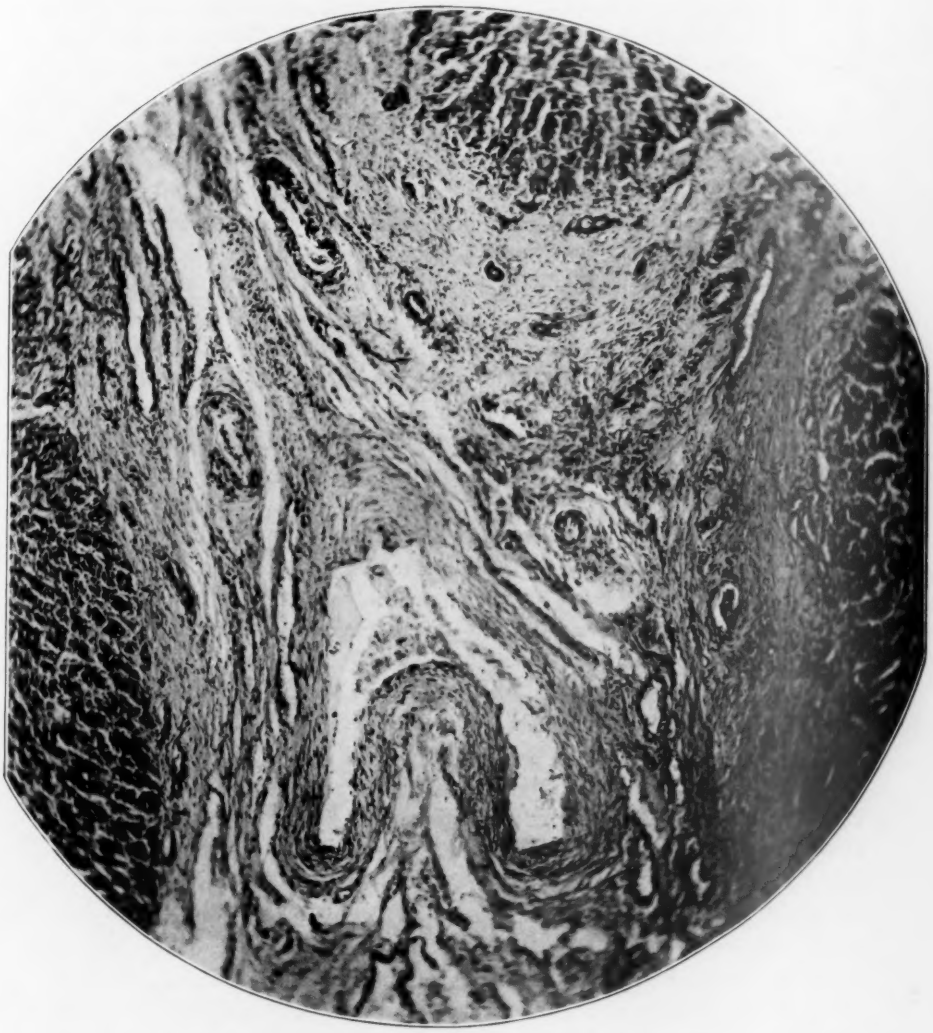
Summary of the Postmortem Examination.—The body was that of an emaciated Negro boy weighing 9 pounds (4,100 Gm.) with the skin and sclerae markedly icteric. The abdomen was greatly distended. The liver was dark green and extended 3 cm. below the xiphoid process and 1 cm. below the right costal margin. The spleen was 2 cm. below the left costal margin. The thymus weighed 11 Gm. The heart weighed 35 Gm., and the myocardium was pale. The lungs were subcrepitant anteriorly and noncrepitant posteriorly. The spleen weighed 35 Gm. and was soft; the suprarenals weighed 3 Gm. The liver weighed 200 Gm., and was soft and dark olive green. The external surface was everywhere slightly granular. On the superior surface it was mottled with small brown-green areas. The section surface was mottled by dark green and lighter brown-green small areas. The latter areas were located in the center of the acini. In addition, there was scattered throughout a network of narrow gray strands, which appeared, as a rule, at the periphery of the lobules. Around the larger bile ducts there were collars of connective tissue. The gallbladder was missing. Its normal site was occupied by fatty and fibrous tissue, forming a triangular depression in the liver 1 by 1 cm. and 3 mm. deep. The apex of the triangle was directed toward the anterior edge of the liver. Cutting into the liver at this point failed to disclose any remnants of gallbladder tissue. No common bile duct could be probed from the papilla of Vater, although the pancreatic duct was present and normally located. Neither the cystic nor the hepatic ducts could be located. The portal vein, the hepatic artery and the hepatic veins showed no gross abnormal changes. The ductus venosus was obliterated. The intestinal contents were light gray, formed and acholic. The lymph nodes were enlarged to 1 cm. The costochondral junction was much widened and knoblike.

Anatomic Diagnosis.—The anatomic diagnosis was as follows (1) complete absence of the extrahepatic bile ducts, including the gallbladder; (2) peribiliary cirrhosis of the liver; (3) icterus gravis; (4) edema of the lungs and (5) severe rickets.

Microscopic Examination.—Hepatoduodenal Ligament: Sections taken from six different levels of the hepatoduodenal ligament between the papilla of Vater and the hilus of the liver failed to reveal large bile ducts or any structures which could be identified with certainty as the remnants. The portal vein was well developed, and the intima showed moderate fibrous thickening. The hepatic artery was unchanged. About the vessels were numerous nerve bundles, ganglions and small lymph nodes, the endothelium of which was proliferated and filled by yellow-brown pigment.

Liver: The periportal tissue was distinctly increased, seldom to the extent of a complete isolation of single lobules. The proliferated periportal tissue contained newly formed bile ducts in considerable number, while the larger bile ducts were distinctly dilated. The architecture of the liver was distorted by areas of atrophy alternating with areas of regeneration. The atrophic areas usually occupied the

centers of the acini, while the regenerated areas were found in the periphery. The atrophic liver cells were small, stained deeply and contained much brown pigment. The intercellular capillaries were plugged by deep green casts. The Kupffer cells were swollen and contained gray-brown and green pigment granules. The



Large blood vessel in the region of the site of the gallbladder; × 100.

pericapillary reticulum was thickened, and this thickening was most marked near the central veins. The regenerated liver cells stained lightly, had distinct membranes, and were finely granular and free from pigment. There was an occasional group of tubular glands with narrow lumen and low cuboidal epithelium which seemed to fuse with the liver cell cords.

Section Through the Gallbladder Site: Corresponding to the groove found grossly, one found a definite depression of Glisson's capsule, at which point it was

markedly thickened by dense fibrous tissue. In this connective tissue there were many proliferated bile ducts adjacent to the parenchyma of the liver. The latter did not seem to be compressed, and the liver cords appeared well preserved. In the V-shaped portion formed by the groove was found a loose fibrous connective tissue in which, occupying a low power field, was a large blood vessel, the wall of which was thick and contained much muscle and elastic tissue. In addition, there were smaller vessels of the arterial and capillary type. No evidence of gallbladder structure was noted (figure).

COMMENT

Embryology.—Perhaps a few words on the embryology of the biliary tract might be of aid in a clearer understanding of the condition. The gallbladder arises from the saccular portion of the extremity of the hepatic diverticulum. The liver ducts connect the diverticulum with the liver cords. One of these persists as the hepatic duct which joins the cystic duct of the gallbladder. The portion of the diverticulum proximal to this union becomes the common duct, or ductus choledochus.[12] The gallbladder and the common duct are solid in the embryo of 6.8 mm. (Piper, 1900) and in that of 6.75 mm. (Kerbel and Elze, 1908). These are the youngest stages of the embryo which have been observed. In the embryo of 7.5 mm. there is a lumen in the common bile duct, but the gallbladder is impervious. Near the hepatic duct the lumen is subdivided, appearing in cross-section as two or three minute pores. In embryos of 10 mm. the gallbladder and the extrahepatic ducts have become longer and more slender. In embryos of 16 mm. there are irregular subdivisions near the hepatic duct, and the distal part of the gallbladder is solid, but in both the cystic duct and the common duct the lumen is single and well defined. Arey [13] stated that the cavity of the gallbladder appears after the embryo is 15 mm. and is surrounded by high columnar epithelium. The extrahepatic ducts are first patent, then lose their lumens by a proliferation of the epithelium to become solid cords, only to become patent later. The complexity of the embryologic development of the liver and bile ducts makes the wide variety of malformations seem logical (Ylppö [14]).

Etiology.—Many theories have been offered as to the possible cause of malformation of the biliary tract: 1. Primary developmental disturbances are manifested by a complete absence of the bile ducts. 2. Primary developmental disturbances cause an abnormal narrowing of the lumens and are followed by a catarrhal condition in the narrowed ducts. The interference in the exit of bile causes a stasis, which disposes to a catarrhal condition, blockage of the passages, and finally an obliteration of the lumens. The portion in which the lumen has been obliterated

13. Arey, L. B.: Developmental Anatomy, Philadelphia, W. B. Saunders Company, 1925, p. 111.

14. Ylppö: Ztschr. f. Kinderh. **60**:317, 1913.

then persists as a fibrous cord or disappears entirely, the obliteration being complete in intra-uterine life.[3] The biliary cirrhosis of the liver which follows is a secondary manifestation. 3. Congenital syphilis causes the malformation. 4. The anomaly originates in fetal peritonitis with the formation of adhesions and constrictions. 5. Toxins, presumably derived from the mother, are excreted by the liver of the fetus and set up in the organ a cholangeitis which subsequently descends to the larger bile ducts and there leads later to obliteration. This view is strengthened by the almost constant finding of cirrhosis in cases of congenital obstruction of the bile ducts as compared with the infrequency and irregularity with which this disease follows obstruction of the larger bile ducts in later life; also, the large size of the liver in congenital cases, and its resemblance in this respect to the liver of hypertrophic biliary cirrhosis. To support the theory of toxins, there are several recorded cases of women having more than 1 child similarly affected, and in Thompson's series there were 2 such cases. Wyard [4] suggested that the condition may be the result of an ascending cholangeitis due to a toxin, a constituent of a pathologic liquor amnii. 6. A mechanical factor might be considered, such as an enormous liver exerting traction on the mesentery or bands of the mesentery, thereby twisting or blocking the common duct. The rapid growth of the liver could twist or constrict the ductus choledochus at its duodenal outlet, tying it off. This mechanical theory has been described by Benecke,[15] as the "Theorie des Abschnürungs-Prozesses" (theory of process of constriction). He supported his theory by his findings in 74 cases; most of them showed a constriction atresia at the level of the papilla of Vater. It is well known that epithelial concrescences take place in the alimentary tract and if the process occurs in the bile ducts it explains the observations of congenital malformations far better than can be done by other theories. An ingrowing of the duodenal epithelium might so overgrow as to block the tube, as might the epithelium of the duodenal end of the duct itself. An inflammatory process radiating from the liver outward might do so, since the bile ducts are so extremely small at birth that any inflammatory change will, from the small size of the lumens, produce a stenosis more readily than in later life. It is possible that abnormalities of the fetal vitelline and umbilical trunks may result in anomalies of the biliary apparatus.[13]

Rolleston and Hayne [2] expressed the belief that the cirrhosis of the liver is the primary condition, and that the lesions in the bile ducts are secondary. Parsons and Hickmans [1] were of the opinion that the intrahepatic process occurs simultaneously with the changes in the extrahepatic bile ducts.

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15. Benecke, R.: Die Entstehung der kongenitalen Artresie der grossen Gallengänge, nebst Bermerkungen über den Begriff der Abschnürung, Universität's Programm Marburg, 1907; quoted by Holmes.[5]

In the case reported here there was no evidence of syphilis, infection, toxin or peritonitis as a possible factor in the etiology of this condition. Hence, in all probability a congenital malformation (vitiums primae formations) was being dealt with just as one may find congenital anomalies in the heart or elsewhere in the body. From the section of the liver corresponding to the normal site of the gallbladder, one might infer that the groove seen microscopically might have been formed by the pressure of a gallbladder from without inward. The groove might also have been produced by the pressure of the hepatic blood vessels at their entrance. The latter, I feel, is worthy of consideration because of the evidence seen microscopically of the presence of large-sized blood vessels where the gallbladder should normally have been.

The histories of the cases of absence of the gallbladder and extrahepatic bile ducts are fairly constant as to a progressive jaundice appearing from a few days to a week after birth, which is persistent to the end. The liver is usually found enlarged. The nutritional state of the child may be good until just before death. The urine is dark, and the stools are light yellow to clay-colored. The expectancy of life may be as much as eight months.

SUMMARY

A case of complete absence of the gallbladder, the hepatic ducts, cystic duct and common duct in a child that lived for five months is described. This absence is explained on the basis of a congenital malformation of the biliary tract. Secondary biliary cirrhosis of the liver due to bile stasis and general icterus were present.

Progress in Pediatrics

HEART BLOCK IN RHEUMATIC FEVER

MAX POMERANCE, M.D.
AND
SIMON FRUCHT, M.D.
BROOKLYN

Acute rheumatic fever, aptly characterized as a disease which "licks the joints and bites the heart," has attracted world-wide attention within recent years. The close association between rheumatic fever and heart disease was noted many years ago by early English writers. The present conception rests on the work of Bouillaud[1] (1836), who singled out the heart as the chief sufferer. He called attention to the permanent nature of the cardiac injury and described alterations in the functional activities of the heart. With the introduction of graphic methods in the physiologic laboratory and at the bedside, an intensive study of the heart took place. Many investigators interested themselves in following up the rheumatic cases in order to observe the myocardial changes. At the same time there appeared at intervals a number of clinical and pathologic reports which led to an astonishing result. It was shown that the first evidence and sometimes the only evidence of myocardial impairment was found in the polygram and the electrocardiogram. It was also demonstrated that the heart was affected in practically 100 per cent of the cases in rheumatic fever. It is unnecessary to dilate on the importance of this discovery. Rheumatic fever and its aftermath are too well known to warrant further comment.

The electrocardiogram presents a fairly constant picture for the normal heart. Deviations from the normal are met with great frequency during the acute stage of a rheumatic attack, and their fleeting character has been the subject of persistent remark. Cardiac irregularities of almost every description have been observed. Changes in axis-deviation, premature beats and every gradation of heart block have been described. The most frequent disturbance has been found to be delayed auriculoventricular conduction, ranging from the simple P R interval prolongation to complete dissociation. Heart block in rheumatism is so characteristic that a presumptive diagnosis of rheumatic fever may be

1. Bouillaud, J. B.: Traité clinique du rhumatisme articulaire et de la loi de coincidence des inflammations du cœur avec cette maladie, Paris, J. B. Baillière, 1840.

made a number of days prior to the onset of the clinical manifestations. Paul D. White [2] reported a case of acute rheumatic fever in which heart block was the first sign, and it preceded all other clinical findings by several days.

The changes in cardiac rhythm and the electrocardiographic alterations met with in acute rheumatic fever are numerous. For convenience, they have been tabulated and arranged according to the site of origin and functional disturbance (tables 1 and 2).

TABLE 1.—*Origin of Disturbances in Cardiac Rhythm*

Site of Origin	Cardiac Disturbance
Sinus node	a. Sinus arrhythmia b. Sinus block
Auriculoventricular node	a. Incomplete block (prolonged P R interval) b. Partial block (auricular dropped beats) c. Nodal premature beats d. Nodal rhythm e. Partial heart block (2 to 1 rhythm) f. Complete heart block g. Auriculoventricular dissociation
Branches of bundle of His	a. Left bundle branch block b. Right bundle branch block
Auricles	a. Auricular premature beats b. Auricular paroxysmal tachycardia c. Auricular flutter d. Auricular fibrillation
Ventricles	a. Ventricular premature beats b. Ventricular paroxysmal tachycardia c. Arborization block

TABLE 2.—*Electrocardiographic Changes Observed in Rheumatic Fever*

Deflection	Electrocardiographic Alteration
P	a. Widening b. Notching
Q R S	a. Notching or slurring b. Widening c. Alteration in amplitude d. Left axis deviation e. Right axis deviation
T	a. Flattening b. Inversion

The outstanding feature of the rheumatic attack is the extent and the frequency of the myocardial involvement. Cohn and Swift [3] made a study of thirty-seven cases of rheumatic fever and found the heart affected in thirty-five. In a separate study of eighty-one rheumatic patients, Swift [4] reported electrocardiographic changes in seventy (86

2. White, P. D.: Acute Heart Block Occurring as the First Sign of Rheumatic Fever, Am. J. M. Sc. **152**:589, 1916.

3. Cohn, A. E., and Swift, H. F.: Electrocardiographic Evidence of Myocardial Involvement in Rheumatic Fever, J. Exper. Med. **39**:1, 1924.

4. Swift, H. F.: Rheumatic Fever, Am. J. M. Sc. **170**:631, 1925.

per cent). Rothschild, Sachs and Libman,[5] in a survey of sixty-five cases of rheumatic fever, discovered electrocardiographic changes in sixty-one (93 per cent). McMillan and Cook[6] reported one or more alterations of the electric curve in thirty-six of forty-three cases of rheumatic fever.

Every cardiac irregularity occurring during an attack of rheumatic fever may be considered as a form of block. The term, heart block, has been reserved for those disorders of rhythm which interfere with the conduction of the excitation wave in its passage through the heart. These "troubles de rythme cardiaque" constitute a group of great interest to the clinician. Tracings of rheumatic patients disclose every known form of block, the variety and degree depending on the severity of the infection.

Heart block in rheumatic fever presents several highly interesting and unusual features. It appears and disappears, at times with a suddenness that is most disconcerting. In this respect, the heart block conforms to some of the clinical aspects of rheumatic fever. The fleeting and frequently unrecognized pains in the joints in children are often a source of mortification to parents as well as to the attending physician. The ephemeral nature of the rheumatic nodule, the transitory pericardial friction rub and the inconstant cutaneous manifestations bear witness to a distinctive characteristic of rheumatic fever, its variability and changeableness. In the electrocardiogram for case 3 (fig. 4) there can be seen three distinct forms of cardiac rhythm, which appeared successively within the few minutes during which the tracing was being registered. On the other hand, when the rheumatic attack has been exceptionally severe, scar tissue replaces the myocardium, and functional disturbances, more or less permanent, are the result. The electrocardiogram for case 1 (fig. 1) shows a left bundle branch block[7] during the acute stage. A tracing taken three months later showed only slight improvement (fig. 2).

In some instances, the heart block disclosed a ventricular rate accelerated to such a point that the diagnosis is rendered impossible by the ordinary methods. The electrocardiogram for case 2 (fig. 3) presents an auricular rate of 120 and a ventricular rate of 80. It is conceivable that cases of complete heart block in rheumatic fever go unrecognized because of a ventricular rate above 70. For an accurate diagnosis the electrocardiogram is indispensable.

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5. Rothschild, M. A.; Sachs, B., and Libman, E.: Disturbances in the Cardiac Mechanism in Subacute Bacterial Endocarditis and Rheumatic Fever, Am. Heart J. **2**:356, 1926.

6. McMillan, T. M., and Cook, A.: Electrocardiographic Changes Occurring During Rheumatic Fever, Atlantic M. J. **31**:806, 1927.

7. Frucht, S.: The Electrocardiogram in Practice, M. Times & Long Island M. J. **69**:383 (Nov.) 1931.

Heart block in rheumatic fever may present a striking dissociation between auricle and ventricle in the form of a curve which shows a ventricular rate greater than the auricular rate. The electrocardiogram for case 3 (fig. 4) exhibits a ventricular rate of 120 and an auricular rate of 108. Of this peculiar rhythm there are few examples in the literature. White [8] (1916) published a record of a young woman, aged 24, who suffered from a peritonsillar abscess four weeks prior to a tonsillectomy under ether anesthesia. An electrocardiogram taken four days after the tonsillectomy showed a complete dissociation in conduction; the ventricular rate was 96, and the auricular rate ranged between 67 and 85. Lea [9] (1915) collected eleven cases of auriculoventricular dissociation, including two of his own, in which the ventricular rate was greater than the auricular rate. McMillan and Cook [6] (1927) described two cases of rheumatic fever in which the ventricular rate was higher than the auricular rate. Published reports of this remarkable type of complete auriculoventricular dissociation are very few, and records of those seen during a rheumatic attack are rare.

The state of the myocardium is a closed book in the routine examination of rheumatic patients. In 1913, Lewis [10] called attention to the effect of the rheumatic invasion on the myocardium. He cited case after case to show the value and the importance of obtaining accurate records by means of the polygram and the electrocardiogram. He stressed the frequency with which heart block appears in rheumatic fever and presented tracings depicting various grades of heart block. The state of the myocardium is revealed in accents unmistakable by the electrocardiogram.

Complete dissociation between auricle and ventricle is an event of serious import. It is rarely encountered in rheumatic fever. A careful review of the literature brought to light isolated reports from widely scattered sources. The earliest record is that of Rihl [11] in 1906. Joachim [12] (1907) described a case of rheumatism in a young man of 24 years, who had a pulse rate of 35. Gill [13] (1909) had two cases of

8. White, P. D.: Ventricular Escape with Observations on Cases Showing a Ventricular Rate Greater than That of the Auricles, Arch. Int. Med. **18**:244 (Aug.) 1916.

9. Lea, E.: Complete Heart-Block with Higher Ventricular Than Auricular Rate, Lancet **1**:1289, 1915.

10. Lewis, T.: Certain Physical Signs of Myocardial Involvement, Brit. M. J. **1**:484, 1913.

11. Rihl, J.: Analyse von fünf Fällen von Überleitungsstörungen, Ztschr. f. exper. Med. u. Therap. **2**:83, 1906.

12. Joachim: Weitere Beiträge zur Frage der Leitungsstörungen im Herzmuskel, Deutsches Arch. f. klin. Med. **88**:574, 1907.

13. Gill: Two Cases of Heart-Block in Acute Rheumatism, Australian M. Gaz. **28**:374, 1909.

rheumatic fever which presented complete heart block. Bramwell [14] (1909) reported a case of heart block in a patient of 29 years, who became ill ten days previously with an attack of rheumatic fever. The polygram was seen by MacKenzie who confirmed the diagnosis of complete heart block. Gerhardt [15] (1909) described a case of complete heart block of rheumatic origin with recovery. Four months later the patient died of typhoid fever. Autopsy showed an infiltration limited to the area about the bundle of His. Magnus-Alsleben [16] (1909) recorded a case of rheumatic fever presenting a pulse rate of 34. Pardee [17] (1913) published a report of a patient in whom complete heart block developed during an attack of rheumatic fever. The patient was an Italian laborer, aged 23, suffering from rheumatism for three months prior to admission to the hospital. His pulse rate on admission was 82, on the third day it fell to 29. Several days later the patient died. Autopsy showed a lesion which had destroyed one half of the transverse section of the auriculoventricular bundle, the earmark of a previous rheumatic attack. Superimposed on this lesion was an extensive diffuse inflammation involving the second half of the bundle of His. The destruction of both halves of the bundle of His gave rise to the complete heart block. Routier [18] and Esmein, Pezzi and Donzelot [19] described rheumatic patients that exhibited complete heart block during the acute stage. A case of complete heart block, rheumatic in origin, was reported by Holst [20] in 1914. Crawford [21] (1927) published his observations on heart block in a patient with rheumatic fever under treatment with digitalis. His patient had a rapid ventricular rate. Carr and Reddick [22] (1928) presented four cases of complete heart block in rheumatic fever. In two instances the ventricular rate was 36. The third patient had an auricular rate of 110 and a ventricular rate of 83. Normal rhythm was

14. Bramwell, B.: A Case of Heart-Block with Fibrous Degeneration and Partial Obliteration of the Bundle of His, Brit. M. J. **1**:995, 1909.

15. Gerhardt: Deutsches Arch. f. klin. Med. **23**:485, 1909.

16. Magnus-Alsleben: Zur Kenntnis der vorübergehenden Ueberleitungsstörungen des Herzens, Ztschr. f. klin. Med. **69**:82, 1909.

17. Pardee, H. E. B.: The Relation of Heart Block to Lesions of the Auriculoventricular Bundle, Arch. Int. Med. **11**:641 (June) 1913.

18. Routier, D.: Dissociation auriculo-ventriculaire transitoire dans le rhumatisme articulaire aigu, Arch. d. mal. du cœur **7**:316, 1914.

19. Esmein, C.; Pezzi, C., and Donzelot, E.: Troubles du rythme cardiaque chez deux malades atteints de rhumatisme articulaire aigu, Arch. d. mal. du cœur **7**:323, 1914.

20. Holst, P. F.: Norsk mag. f. lægevidensk. **5**:1369, 1914.

21. Crawford, J. H.: Observations on a Case of Heart Block in Rheumatic Fever Under Treatment with Digitalis, Am. Heart J. **3**:196, 1927.

22. Carr, J. G. ,and Reddick, W. G.: Conduction Disturbances in Acute Rheumatic Fever, J. A. M. A. **91**:853 (Sept. 22) 1928.

restored after eight days. For the fourth patient, a girl of 9 years, an auricular rate of 136 and a ventricular rate of 71 were registered. Twelve days later, an electrocardiogram showed normal rhythm. Castex, Lopez and Repetto [23] (1928) recorded two cases of rheumatic fever with complete heart block. The authors stressed the transitory nature of the heart block. Vialard [24] (1928) included a case of complete heart block in a paper devoted to the electrocardiographic changes incident to acute rheumatic fever. Padilla and Cossio [25] (1930) reported three cases of complete heart block in rheumatic fever during the acute attack. They also emphasized the transient character of the cardiac disturbance. Bloom and Perlow [26] (1930) described two cases of complete heart block with accelerated ventricular rates. One of the patients suffered from rheumatic arthritis. Campbell [27] (1931) published an account of a rheumatic patient in whom complete heart block developed. The auricular rate was 114 and the ventricular rate, 62. Lian and Calcena [28] (1932) made a study of twenty-eight cases of rheumatic fever. In one a complete heart block developed which lasted for two days. The heart block was not diagnosed clinically because of the rapid ventricular rate. Ellis [29] (1932), in a survey of twenty-nine cases of complete heart block, found two that occurred in rheumatic fever. Salvesen [30] (1932) described a patient, 30 years old, who had an infection that was diagnosed as influenza. The electrocardiogram showed a complete heart block with a ventricular rate of 70. The complete heart block lasted for two days. It was succeeded by a cardiac rhythm which showed varying degrees of partial heart block. The presence of the heart block changed the diagnosis to acute rheumatic fever. Our own case was that of a girl, aged 14, who suffered from a fatal rheumatic carditis. The electrocardiogram showed a complete heart block with a ventricular rate of 80 (fig. 3).

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23. Castex, M. R.; Lopez, R. R., and Repetto, R. L.: Dos casos de blocqueo a. v. completo, de origen reumatico, Rev. Soc. de med. int. y Soc. de tisiol. **4**:261, 1928.

24. Vialard, R.: Les troubles du rythme cardiaque dans le cours du rhumatisme articulaire aigu, Thèse de Paris, 1929.

25. Padilla, F., and Cossio, P.: Alteraciones de la conductibilidad de origen reumatico, Semana méd. **2**:1469 (Nov. 13) 1930.

26. Bloom, B., and Perlow, S.: Complete Heart Block Associated with Rapid Ventricular Rate, Am. Heart J. **5**:486, 1929.

27. Campbell, M.: Complete and Partial Heart-Block During an Attack of Rheumatic Fever, Lancet **2**:180 (July 25) 1931.

28. Lian, C., and Calcena, B.: Les troubles myocardiques révélés par l'électrocardiographie pendant et après le rhumatisme articulaire aigu, Presse méd. **40**:1 (Jan. 2) 1932.

29. Ellis, L. B.: Studies in Heart Block, Am. J. M. Sc. **183**:225 (Feb.) 1932.

30. Salvesen, H. A.: Complete Heart Block in a Case of Acute Rheumatic Infection, Acta med. Scandinav. **78**:189, 1932.

REPORT OF CASES

CASE 1.—*History.*—K. H., a boy, aged 9 years, was admitted to the surgical service of Dr. Iason on March 26, 1931, because of abdominal cramps of four days' duration. The patient had had three attacks of pneumonia. He was informed of a cardiac involvement by the school physician. The physical examination revealed rheumatic pancarditis, and the patient was transferred to the service of Dr. H. Ratnoff.

Examination.—The patient was acutely ill. There was marked pallor, the pharynx was congested, and the pulse rate was regular and rapid (144). The heart was enlarged to the right and left, and there were a presystolic thrill at the apex, muffled heart sounds, a presystolic murmur and systolic and diastolic murmurs at Erb's point. There was dulness in the lungs, with bronchial breathing and bronchophony over the left side of the chest posteriorly and subcrepitant and

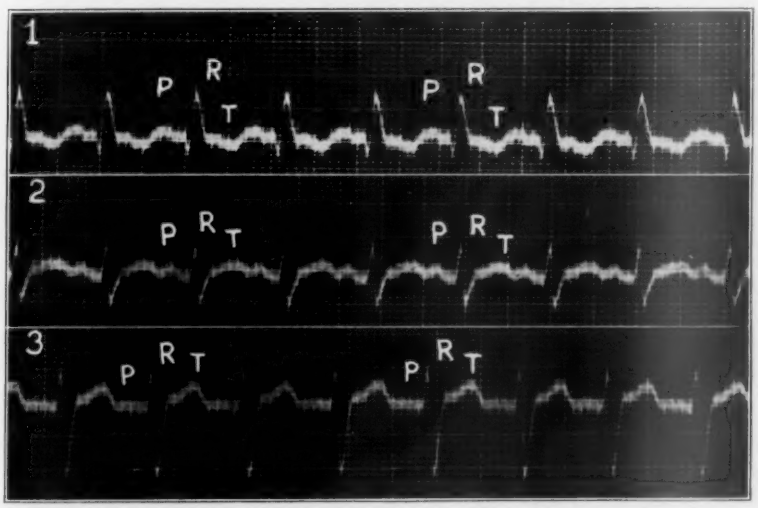

Fig. 1 (case 1).—Electrocardiogram taken on April 15, 1931, showing left bundle branch block. R in lead I and S in lead III, T in the opposite direction to the main deflection and a widened Q R S complex indicate a left bundle branch block.

crepitant râles at the base of the right lung. The blood count showed: white cells, 15,800; polymorphonuclears, 84 per cent. A blood culture was negative. There was a trace of albumin in the urine. Roentgen examination showed the cardiac shadow markedly widened, especially to the right; the cardiac shadow extended 6.3 cm. to the right of the midsternal line and 8.4 cm. to the left; the internal transverse thoracic measurement was 21.2 cm., and there was mottling at the base of the right lung, with some obscuration of the right leaflet. The electrocardiogram, taken on April 15, showed a left bundle branch block (fig. 1).

The condition was diagnosed as recurrent rheumatic endocarditis affecting the mitral and aortic valves, pericarditis with effusion and a left bundle branch block.

Course.—The patient remained in the hospital for five weeks. His temperature ranged from 99 to 104 F. He was given salicylates and digifolin (1 minim

[0.18 Gm.] every four hours). The digifolin was discontinued on April 1. He was discharged as improved on April 30.

An electrocardiogram taken July 18 showed a slight improvement. The Q R S widening was somewhat diminished and the inversion of T in lead I was less pronounced (fig. 2).

CASE 2.—*History.*—F. W., a girl, aged 14 years, was admitted to the service of Dr. L. Harris at Beth-El Hospital on May 25, 1931, because of pain over the heart and shortness of breath. She had had measles, whooping cough and chicken pox, and seven years before admission, bronchopneumonia. Six years before she had had rheumatic fever, with almost all the joints involved, and had been ill for sixteen weeks. Five years before she had had a second attack of rheumatic fever, at which time purpuric spots were noted, and had been ill for four months. One

Fig. 2 (case 1).—Electrocardiogram taken on July 18, 1931, showing left bundle branch block. A comparison with figure 1 shows the Q R S complex diminished in width and the inversion of T in lead I less pronounced.

year before she had had a third attack of rheumatic fever which lasted for three months. Thirteen weeks before admission, the patient had had pains in the joints and a sore throat. She was in bed for several weeks, and felt better until nine weeks before admission, when a recurrence of the pains in the joints required hospitalization for three weeks. Five days before admission she had had a similar attack with swelling of the feet.

The patient appeared to be acutely ill. There were pallor and cyanosis, a regular pulse (rate 80), a systolic carotid thrill and a heaving apex beat. The heart was enlarged to the right and left, with the left border at the anterior axillary line. There was a short presystolic murmur at the apex and a systolic and diastolic murmur at Erb's point. There were râles at both bases. The liver was palpable four fingerbreadths below the costal margin; the spleen was not palpable. There

was edema of the lower extremities. Laboratory tests showed: red cells, 4,350,000; white cells, 18,000; polymorphonuclears, 78 per cent. A blood culture was negative and the blood chemistry, normal. Urinalysis revealed albumin, 2 plus. Roentgen examination showed a heart of the ovoid mitral type, considerably enlarged to the right and left and straightened at the left auricular border; the cardiac measurements were 4 cm. to the right of the midsternal line and 11 cm. to the left, and the internal transverse thoracic measurement was 22 cm. An electrocardiogram, taken on May 26, showed a complete heart block (fig. 3).

The condition was diagnosed as acute rheumatic fever with complete heart block and cardiac decompensation.

Course.—The patient was in the hospital from May 25 to June 12. Her temperature ranged from 100 to 104 F. and respirations from 30 to 40. The cyanosis deepened, the edema spread, coma developed, and the patient died on June 12.

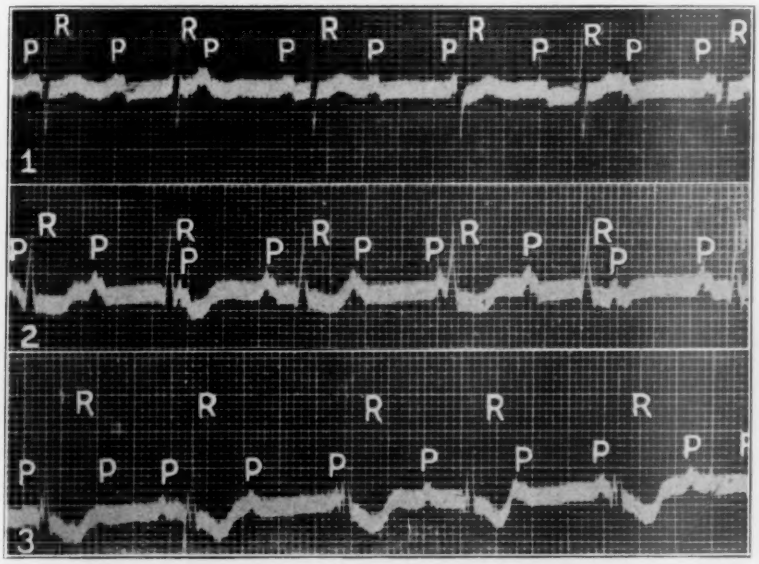

Fig. 3 (case 2).—Electrocardiogram taken on May 26, 1931, showing complete heart block. The auricular rate is 120; the ventricular rate, 80. S in lead I and R in leads II and III indicate a tendency to right axis deviation. The notching of P in leads I and II indicates auricular enlargement as seen in mitral stenosis. Inversion of T in leads II and III indicates myocardial changes at the apex.

Medication included: codeine sulphate, ¼ grain (0.016 Gm.) three times a day, discontinued on May 31; digitalis, 1½ grains (0.1 Gm.) three times a day, changed to digifolin ampules three times a day, on June 8; sodium salicylate, 10 grains (0.65 Gm.), three times a day, changed to 60 grains (3.9 Gm.) by rectum three times a day on June 11; salyrgan, administered intravenously on June 12.

Case 3.—*History.*—S. G., a boy, aged 3½ years, was admitted to the service of Dr. H. Ratnoff at the Beth-El Hospital on April 17, 1931, because of pains in the joints, shortness of breath and vomiting. The history was irrelevant except for frequent sore throat. Four weeks prior to admission there developed redness and swelling of the joints of the ankles, which gradually spread to the knees, elbows

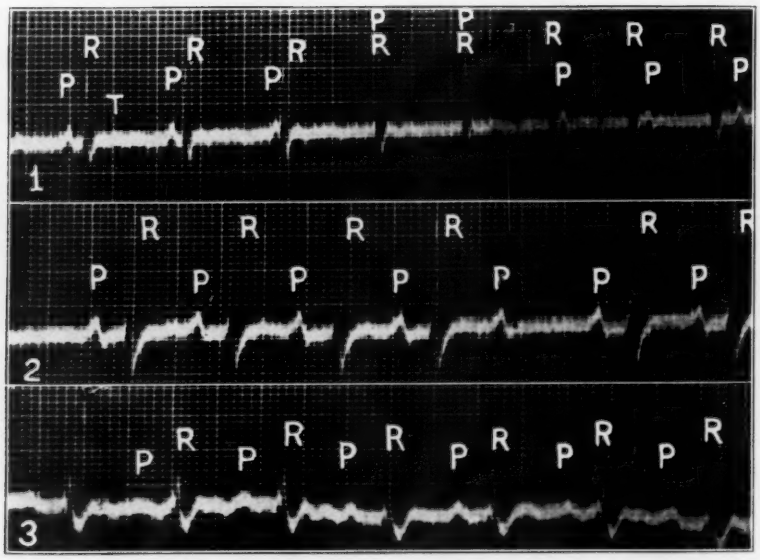

Fig. 4 (case 3).—Electrocardiogram taken on April 17, 1931, showing auriculoventricular dissociation. In lead I there is a complete auriculoventricular dissociation, with the ventricular rate higher than the auricular rate (ventricular rate, 120; auricular rate, 108). In the first cycle, the P R interval is 0.12 second. The P R interval decreases progressively till P becomes superimposed on R in the fourth cycle. In the succeeding cycles P follows R, the P R interval increasing progressively. In lead II there is a blocked auricular wave P in the fifth cycle, indicating a partial block. In lead III there is a prolongation of the P R interval (0.24 second) indicating an incomplete block. The three abnormal rhythms were found during the few minutes it took to register the tracing.

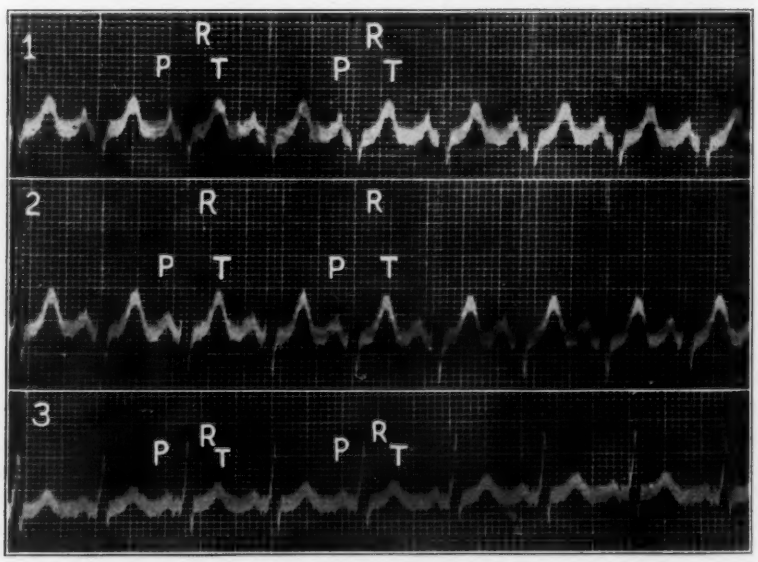

Fig. 5 (case 3).—Electrocardiogram taken on July 15, 1931, showing the normal rhythm restored.

and shoulders. The temperature varied from 100 to 104 F. This condition lasted two weeks, following which the child had loss of appetite, vomiting and shortness of breath. Examination showed a poorly nourished child who appeared to be acutely ill. The tonsils were enlarged and the throat was congested. The heart was enlarged to the left, with a heaving apex beat, a loud systolic murmur at the apex and systolic and diastolic murmurs at Erb's point. The lungs were clear and the abdomen normal. The blood count showed: red blood cells, 3,200,000; white blood cells, 10,800; polymorphonuclears, 74 per cent; lymphocytes, 26 per cent; hemoglobin, 62 per cent. The urine was normal. Roentgen examination showed the cardiac shadow enlarged in all directions; the cardiac shadow extended 3.7 cm. to the right of the midsternal line and 6.6 cm. to the left, and the internal transverse thoracic measurement was 18.5 cm. An electrocardiogram, taken April 17, showed auriculoventricular dissociation, incomplete block and partial heart block (fig. 4).

The condition was diagnosed as acute rheumatic fever and rheumatic endocarditis involving the mitral and aortic valves.

Course.—The patient was in the hospital for two weeks. The temperature varied from 99.6 to 101 F., the respiratory rate from 28 to 40 and the pulse rate from 128 to 140. Sodium salicylate was administered in doses of 5 grains (0.32 Gm.) three times a day. The patient was discharged as improved on April 30.

An electrocardiogram, taken on July 15, showed a normal rhythm (fig. 5).

COMMENT

The electrocardiographic changes in acute rheumatic fever are the result of blocking the normal spread of the excitation wave in a given area of the heart. The form of the block depends on the extent of the injury and the region affected. In this paper we have been particularly interested in the complete dissociation between the upper and lower chambers of the heart. Complete heart block is produced by a lesion within and about the auriculoventricular bundle, as seen in the classic report of Lewis [31] on the autopsy in Dr. H. J. Starling's case of heart block. Lewis found a calcareous nodule situated exactly in front of the bifurcation of the bundle of His, both branches of which were invaded by a chronic inflammatory process resulting in a complete break in conduction. In Gerhardt's case of complete heart block, the lesion was limited to the vicinity of the auriculoventricular bundle. In the case of Pardee, a recent diffuse inflammation was saddled on a previous lesion which had destroyed one half of the bundle of His. The two episodes brought on a complete heart block.

More pertinent to our inquiry is the reason for the frequent appearance of heart block in acute rheumatic fever. In our own series of rheumatic cases, thirty-one in number, twenty-eight cases showed prolongation of the P R interval (incomplete block). The other three cases, showing complete dissociation, complete heart block and left bundle

31. Lewis, T.: Post Mortem Notes of Dr. H. J. Starling's Case of Heart-Block, Heart **9**:284, 1911.

branch block, are reported here. The answer to the problem is found in the curious predilection of the rheumatic infection for the tissues immediately surrounding the bundle of His. Wiggers [32] explained the heart block on the anatomic structure of the auriculoventricular node, which lies embedded in a synovial sac. "Fluid accumulating in this unyielding structure acts by compression to produce an incomplete, then a partial, and more rarely a complete heart block. As the fluid is reabsorbed, the block disappears by reverse steps." In case 3 is the exact reproduction of the process outlined by Wiggers. Lead I shows complete dissociation, lead II partial heart block and lead III incomplete heart block. It is possible that the electrocardiogram was being taken at the time that resorption was taking place. Lewis [10] pictured the invasion of the rheumatic infection as an exudation of leukocytes attended by engorgement and tumefaction. As the inflammation subsides, the fluid is reabsorbed and the tension on the auriculoventricular bundle is relieved. Aschoff and Tawara [33] described the rheumatic attack as an interstitial myocarditis characterized by the presence of small round nodules, the Aschoff bodies, which lie in close proximity to a small coronary branch. Coombs [34] found a proliferation of the endothelial cells of the smaller blood vessels and ascribed the cardiac disturbances to circulatory impairment. During the acute stage, the cells encroach on the lumen of the arteriole, shutting off the circulation for the time. The ischemia results in delayed conduction, which in turn gives rise to varying degrees of heart block. With the passing of the acute inflammation, the circulation becomes reestablished, and normal rhythm is restored.

One of us [7] contends that the edema about the Aschoff body during the acute process compresses the fine coronary branch lying adjacent. When the edema clears, the caliber of the arteriole returns to normal, the ischemia disappears and the cardiac irregularities vanish. Circulatory embarrassment, however, cannot account for the singular acceleration of the ventricular rate in complete heart block. A toxic factor must be present in the infection, which acts as an irritant to the ventricular musculature. Impaired circulation in conjunction with toxemia is probably responsible for the vagaries in conduction prevalent in acute rheumatic fever.

The electrocardiogram in case 1 has been interpreted as a left bundle branch block because the Q R S complex is directed upward in lead I. It is our belief that an upright deflection in lead I indicates a resultant

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32. Wiggers, C. J.: Principles and Practice of Electrocardiography, St. Louis, C. V. Mosby Company, 1929, p. 198.

33. Aschoff, L., and Tawara, S.: Die heutige Lehre von den pathologisch-anatomischen Grundlagen der Herzschwäche, Jena, Gustav Fischer, 1906.

34. Coombs, C. F.: Rheumatic Heart Disease, New York, William Wood & Company, 1924, p. 52.

flow of current from right to left.[35] When the direction of the current is from left to right, as in dextrocardia, the deflections become inverted in lead I. We have also been impressed with the striking similarity of the curve of bundle branch block to the form of the ventricular premature beat. As a matter of fact, bundle branch block may be considered as a continued series of ventricular premature contractions originating in an upper, middle or lower third of a particular bundle branch. In that event, the curve of the bundle branch block should be interpreted according to the experiment of Wilson [36] on the exposed human heart. From this point of view, the electrocardiogram in case 1 (fig. 1) is indicative of a left bundle branch block, the site of origin for the ventricular complex being at the base of the right ventricle or in the upper third of the right branch of the bundle of His.

SUMMARY AND CONCLUSIONS

Three cases of acute rheumatic fever are presented as a study in the disturbances of cardiac rhythm occurring during the acute attack. They illustrate four important considerations in the care of rheumatic patients: (1) the fleeting character of the abnormal rhythm; (2) the accelerated ventricular rate in complete heart block, making the clinical diagnosis impossible; (3) the curious auriculoventricular dissociation, in which the ventricular rate is higher than the auricular rate; (4) the necessity for exact records in all rheumatic cases.

1493 Eastern Parkway.

35. Frucht, S.: A Critical Study of the T Wave in the Electrocardiogram, New York State J. Med. **28**:726 (June 15) 1928; Lead 4 of the Electrocardiogram, M. Times & Long Island M. J. **61**:232 (Aug.) 1933.

36. Wilson; Barker; Macleod, and Alexander: The Excitatory Process Observed in the Exposed Human Heart, Tr. A. Am. Physicians **44**:125, 1929.

Progress in Pediatrics

EVALUATION OF NONORGANIC AUSCULTATORY CARDIAC FINDINGS AND THE VENOUS HUM IN CHILDREN

WALTER J. SIEMSEN, M.D.
CHICAGO

A statement made by Sir James Mackenzie [1] in 1925 is not without applicability even today. It reads as follows:

> While we justly acclaim Laënnec for his discovery of auscultation it must be admitted that its introduction into medicine has not only hampered the progress of knowledge of heart affections, but has done more harm than good in that many people have had the tenor of their lives altered, have been forbidden to undertake duties for which they were perfectly competent, and have been subjected to unnecessary treatment. This is not due to auscultation as such, but to the inability of the profession to assess the value of the phenomena which auscultation revealed.

It is in the hope of furthering the eradication of the "evil" referred to by Mackenzie [1] that this article was written.

Because of the impressibility of children the handicap imposed on a child with a normal heart by sentencing him to "cardiac invalidism" may be of greater psychic than physical moment. The child is likely to become introspective and despondent, and oversolicitousness on the part of the parents tends to lead to behavior difficulties. It is a matter of everyday experience that the sick (real or false) child is often a problem ("spoiled") child.

Healthy boys attending the University of Chicago Laboratory Schools and ranging in age from 5 to 18 years served as subjects for the observations reported. For obvious reasons school children approach the normal more closely than any other group. Except for the exclusion of children with abnormal cardiac findings or with physical incapacitation and except for the omission of cases because of incomplete data the material was unselected. Observations were made with the subject standing during the routine annual examinations, the boys thus being unaware of any unusual procedures.

ARRHYTHMIAS

Strictly speaking, the term arrhythmia denotes irregularity in force as well as in rate. However, unless the force of the heart beat is

From the School of Education of the University of Chicago.

1. Mackenzie, J.: Diseases of the Heart, ed. 4, New York, Oxford University Press, 1925.

markedly irregular, the irregularity is not readily detected on auscultation. Irregularity in rate, on the other hand, is a more obtrusive phenomenon. Arrhythmias of cardiac rate are frequent and of various types during childhood. Friberger [2] found that of 321 children from 5 to 14 years of age whom he examined none had an absolutely regular pulse; the majority showed a variation of 0.2 or 0.3 second in the duration of cardiac cycles. Differences as great as this should be detectable even by the inexperienced ear. These variations in the duration of cardiac cycles occur at the expense of the duration of diastole. Hecht [3] found that in some cases it is possible to determine by ear that the interval between the first and second heart tones does not vary but that the interval between the second and the next first sound varies. Electrocardiographically differences much smaller can, of course, be measured accurately. Seham [4] stated that by this method a difference of 0.1 second may be considered evidence of a sinus arrhythmia.

Sinus (nodal, phasic) arrhythmia is by far the most frequent obvious arrhythmia. It results from influences acting via the vagus nerve on the sinus node which determines the rate of impulse formation. This influence can be demonstrated experimentally by the use of atropine, which paralyses the vagus nerve and leads to the disappearance of sinus arrhythmia. Respiration is most prominent among the factors influencing the sinus node via the vagus nerve.

The arrhythmia produced by the influence of respiration is spoken of as respiratory or physiologic arrhythmia. It is synchronous with the phases of respiration (increase in rate during inspiration, decrease during expiration). The term "physiologic arrhythmia" seems entirely justified because of the frequency of this type of irregularity and because of its occurrence in animals as well as in man (Hecht [3]). Mackenzie [1] has gone a step further than to call this type of arrhythmia physiologic; he regards it as being of favorable prognostic significance in organic disease of the heart. Respiratory arrhythmia in children becomes more frequent with advance in age (and a concomitant decrease in cardiac rate). Thus Mackenzie [1] employed the term "youthful irregularity."

While the large majority of rhythmic arrhythmias are synchronous with respiration, there are exceptions. Friberger [2] contended that although a relation between respiration and arrhythmia is recognizable

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2. Friberger, R.: Ueber Arrhythmie bei gesunden Kindern, Arch. f. Kinderh. **58**:30, 1912.

3. Hecht, A. F.: Der Mechanismus der Herzaktion im Kindesalter, seine Physiologie und Pathologie, Ergebn. d. inn. Med. u. Kinderh. **11**:324, 1913.

4. Seham, M.: Electrocardiography in Children, in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 4, p. 282.

in all cases, it may be disturbed or eliminated in many ways. Wiggers [5] stated that slowing is not always synchronous with expiration. Mackenzie [1] described a "periodic variation in rhythm," apparently insignificant, which is similar to the youthful type of irregularity but is independent of respiratory movements. This rhythmic arrhythmia, asynchronous with respiration, was rarely observed in the boys under consideration.

The nonpathologic nature of the arrhythmias described will hardly be challenged by any one. There is, however, a type of irregularity, namely, the extrasystole, about which there is still no unanimity. Differences of opinion are pointed out in articles such as those by Peiper,[6] Leffkowitz,[7] and Antell.[8] Varying prognostic significance is ascribed to extrasystoles according to their point of origin (auricular, auriculoventricular, ventricular), but even in that matter there is disagreement. Most of the unfavorable prognostic views are found in older writings. The tenor has been modulated considerably, and contemporary writers are inclined to regard extrasystoles with much less alarm. Mackenzie,[1] who observed patients with extrasystoles for over forty years, concluded "that when an extrasystole is the only abnormal sign the prognosis is a favorable one; and where it is associated with other signs the prognosis is to be based on these other signs." Other authors, such as Peiper,[6] Leffkowitz,[7] and Antell,[8] share this view.

The incidence of extrasystoles is difficult to appraise, and their etiology is often problematic. The variability of extrasystoles from one examination to another and even during one examination renders statistics as to their incidence practically valueless. According to Mackenzie [1] they are less frequent in children than in adults. The numerous factors which have been cited as causes for extrasystoles can be summarized by the classification of Bass: [9] (1) toxic, (2) emotional and (3) idiopathic. According to Peiper,[6] position (upright or prone) is important.

Extrasystoles were encountered in 3 of the boys examined, aged 10, 11 and 14 years. In 1 instance extrasystoles occurred in showers and in bigeminal form. According to Hecht [3] the bigeminal characteristic

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5. Wiggers, C. J.: Physiology of Circulation, in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 4, p. 198.

6. Peiper, A.: Unregelmässige Herztätigkeit im Kindesalter, Deutsche med. Wchnschr. **56**:1079 (June 27) 1930.

7. Leffkowitz, M.: Ueber Extrasystolen im Kindesalter, Klin. Wchnschr. **10**: 1577 (Aug. 22) 1931.

8. Antell, L.: Premature Contractions of the Heart in Children, Arch. Pediat. **48**:640 (Oct.) 1931.

9. Bass, M. H.: The Significance of Cardiac Extrasystoles in Childhood, J. A. M. A. **86**:387 (Feb. 6) 1926.

is regarded by some authors as belonging to the signs of too long continued digitalis therapy. Merklen [10] declared it a serious finding. The boy manifesting this phenomenon had not received medication and was apparently in good health. Interestingly, the father related that he had observed "skipping" of the pulse at the wrist in himself. The occurrence of extrasystoles in the father lends support to the belief of Antell [8] that heredity plays a rôle in idiopathic cases of extrasystoles. In the other 2 cases exercise seemed to be of excitatory influence. Smith and Sutton [11] stated that extrasystoles may be stopped or induced by exercise, and, according to Peiper,[6] they may or may not disappear after exercise. On subsequent examination 1 of the boys showed extrasystoles; the other 2 did not. The boys were in good health; they showed no signs of organic disease of the heart, and none of them had subjective sensations referable to the extrasystoles. The absence of symptoms, in contrast to the findings in adults, has been stressed by other authors. None of the 12 children observed by Peiper [6] were conscious of extrasystoles. Leffkowitz [7] reported that 2 of 26 children with extrasystoles had subjective symptoms.

THE THIRD HEART SOUND

In addition to the two universally recognized heart sounds, the characteristics of which during childhood are probably too well known to warrant discussion here, a third heart sound is heard at times. According to Smith and Sutton,[11] Gubergritz,[12] and Thayer,[13] this sound is audible in most children if special pains are employed to detect it. However, under ordinary conditions it is heard only occasionally and inconstantly. Observations revealed a third heart sound in about 2 per cent of the boys examined. The sound is short, low-pitched, very soft and heard during diastole at, or just within, the cardiac apex. A slow cardiac rate favors its detection. It is a physiologic phenomenon the genesis of which is not well understood. As pointed out by Wolferth and Margolies,[14] the rhythm produced by three heart tones may simulate protodiastolic gallop rhythm so closely that differentiation is possible only by the absence of cardiac disease in case the rhythm is attributable to three normal heart tones. Gubergritz [12] stressed the difference between the three heart tone rhythm and that produced by split heart tones.

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10. Merklen, quoted by Hecht.[3]

11. Smith, C. H., and Sutton, L. P.: Heart Disease in Infancy and Childhood, New York, D. Appleton and Company, 1930, vol. 18.

12. Gubergritz, M. M.: Ueber gespaltene und sogenannte ueberzählige Herztöne, Ztschr. f. Kreislaufforsch. **21**:65 (Feb.) 1929.

13. Thayer, quoted by Wiggers.[5]

14. Wolferth, C. C., and Margolies, A.: The Various Types of Extra Heart Sounds, M. Clin. North America **14**:897, 1931.

SPLIT (DUPLICATED) HEART SOUNDS

Splitting or duplication of the "standard" two heart sounds is frequent and at times well marked. When the work here reported was first begun, note was made of splitting or duplication. It was soon obvious, however, that the condition is a well-nigh constant finding. Split or duplicated heart sounds consist of two components. Splitting of the first sound, commonly ascribed to dissociation of the sound into its muscular and valvular components or asynchronous contraction of the two ventricles, is heard loudest over the apex. It may be so pronounced as to give rise to a visible biphasic apex impulse and to a simulated or real thrill on palpation. Splitting of the second sound is heard loudest over the base of the heart to the left of the sternum. Pogány [15] placed the point of maximum intensity at the third or fourth left interspaces, the area overlying the conus pulmonalis. The splitting is assumed to result from a difference in the time of closure of the aortic and pulmonic valves, which difference in turn is ascribed to differences in pressure in the systemic and pulmonary circulations. It is conceivable that two mechanisms (a dissociation of the muscular and valvular elements causing a split first sound, and an asynchrony of closure of the aortic and pulmonic valves, due to differences in pressure in the two circulations, causing a split second sound) may operate simultaneously and independently; hence the assumption of Wolferth and Margolies [14] that splitting of both tones is an evidence of asynchronous contraction of the two ventricles, while splitting of the first sound denotes only a delay in closure of the auriculoventricular valves, is probably not requisite. These authors mentioned the difference in the nature of the two elements of a split first sound, the second part usually being sharper and higher-pitched than the first, in support of the contention that the splitting of the first sound is due to a dissociation of the muscular and valvular components. Replacement of the second element of a split first sound by a murmur also favors the view that the first part of a split first sound is muscular and the second part valvular. Wolferth and Margolies [14] have called attention to the fact that while the time interval between the two elements of a split first sound may be so long as to produce a rhythm similar to presystolic gallop rhythm, it may be so short as to be unrecognizable as a split sound but is then heard as a prolonged first sound. "Impure," "rough," "uneven" and "slapping" are other terms used to describe such sounds. Wiggers [5] stated that between the ages of 5 and 15 years vibrations associated with the first sound may produce an impurity of this sound or a functional murmur.

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15. Pogány, J.: Die Spaltung des diastolischen Herztones, Ztschr. f. klin. Med. **116**:206, 1931.

FUNCTIONAL HEART MURMURS

The subject of functional murmurs has formed the basis for an almost unlimited discussion. In accordance with the rule, the tremendous bulk of literature evidences the interest and importance of the subject on the one hand and the incomplete knowledge on the other. Publications have not been limited to medical literature; thus, an informative article by Weinfeld [16] appeared in *Hygeia.*

Widespread interest in functional murmurs is not surprising when one considers the frequency with which they are encountered at all ages, especially during childhood. Figures of older authors giving an incidence well over 50 per cent were cited by Hochsinger,[17] who regarded them as too high. Twenty-seven (8.3 per cent) of 325 school

TABLE 1.—*Incidence of Murmurs Before and After Exercise at Different Ages*

Age	Number of Cases	Murmurs Before Exercise		Murmurs After Exercise	
		Number	Per Cent	Number	Per Cent
5	10	1	10	7	70
6	17	4	24	7	41
7	14	1	7	9	71
8	24	2	8	12	50
9	16	1	6	9	56
10	18	1	6	9	50
11	15	1	7	9	60
12	14	..	..	7	50
13	21	1	5	12	60
14	26	3	12	12	46
15	34	4	12	9	26
16	32	5	16	10	31
17	22	..	..	9	41
18	12	..	..	5	42
Total	275	24	9	126	46

children observed by Lincoln [18] showed functional murmurs at one time or another. On routine examination of patients in the office Gibbes [19] found an incidence of 11 per cent during the first decade of life and of 10.7 per cent during the second. He reported a preponderance for the female sex. The observations recorded in table 1 show an incidence of 9 per cent for boys from 5 to 18 years of age. The relatively high incidence during puberty (fourteenth, fifteenth and sixteenth years) seems significant. Excess strain placed on the heart at this period of life may account for this finding. No explanation can be offered for the high figure for the sixth year.

16. Weinfeld, G. F.: Heart Murmurs in Children, Hygeia **10**:344 (April) 1932.

17. Hochsinger, C.: Ueber bedeutungslose Geräusche in der Präcordialgegend von Kindern und Jugendlichen, Arch. f. Kinderh. **60**:377, 1913.

18. Lincoln, E. M.: The Hearts of Normal Children: I. Clinical Studies, Including Notes on Effort Syndrome, Am. J. Dis. Child. **35**:398 (March) 1928.

19. Gibbes, J. H.: Heart Murmurs: Their Incidence and Interpretation, Am. Heart J. **4**:305 (Feb.) 1929.

While it is readily conceded that the term "functional" as applied to nonpathologic heart murmurs is inappropriate, its continued use is perhaps justifiable so long as no satisfactory term is found. Other terms, as "accidental," "accessory" and "atonic," are used synonymously with the term "functional." The terms "physiologic" and "nonpathologic" are open to the criticism that while a phenomenon may be inconsequential or insignficant, a pathologic genesis is not necessarily precluded. The terms "hemic" and "anemic" have justly been discarded almost entirely. Bass [9] suggested discarding the term "functional" and substituting terms like "atonic," "hemic," "cardiopulmonary" or "accidental" to indicate the cause of murmurs. Unfortunately, the cause of a murmur is frequently obscure.

Explanations regarding the etiology of functional murmurs are largely conjectural. The "cardiorespiratory" murmur is supposedly of extracardiac origin. Griffith and Mitchell [20] were doubtful regarding the possibility of a sharp differentiation between this type of murmur and that of endocardiac origin. They expressed the opinion that most functional murmurs are not cardiorespiratory. Among 27 cases of functional murmurs Lincoln [18] found 1 to be of the cardiorespiratory type. Findings on the boys under consideration favor a low incidence of cardiorespiratory murmurs. While some authors ascribe functional murmurs to a lowered tonicity of the heart and papillary muscles, myogenic or vagal in origin (Epstein [21]), and consequent valvular insufficiency (Schlieps,[22] Smith and Sutton,[11] Griffith and Mitchell [20]), Sawitzki [23] contended that insufficiency of the valves is due to increased tonicity of the papillary muscles. He attributed this condition to a dysfunction of the vegetative nervous system and based his opinion on the disappearance of murmurs after the administration of atropine. He stated his belief that the murmurs are mitral in origin. The fact that many murmurs heard over the mitral area replace only the second (valvular?) part of a split first heart sound favors this view. While many functional murmurs, no doubt, originate at the mitral valve, perhaps a larger number do not, as the favored location of these murmurs is over the base of the heart. As a rule, functional murmurs are soft and blowing (the German term "hauchend" is rather descriptive). They may, however, be low-pitched and harsh or high-pitched and musical. Frequently the murmur is about equally intense over the

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20. Griffith, J. P. C., and Mitchell, A. G.: Diseases of Infants and Children, ed. 2, Philadelphia, W. G. Saunders Company, 1927, vol. 2.

21. Epstein, J.: Myatonic Cardiac Murmurs, Arch. Pediat. **48**:635, 1931.

22. Schlieps, W.: Ueber pseudokardiale und kardiale Geräusche im Kindesalter ohne pathologische Bedeutung, Jahrb. f. Kinderh. **76**:247, 1912.

23. Sawitzki, N. N.: Ueber die Genese des akzidentellen systolischen Herzgeräusches, Ztschr. f. Kreislaufforsch. **21**:6 (Jan.) 1929.

apex and base. The intensity of a murmur may vary during one examination or from one examination to another. It may disappear and reappear. Inconstancy and variability strongly favor the belief that the murmur is functional. While a functional murmur may be heard over the entire precordium, its audibility is usually circumscribed, and it is transmitted poorly or not at all. The time of functional murmurs is almost invariably systolic. Hochsinger,[17] Gibbes,[19] and Chapin and Royster [24] contended that none are diastolic.

In the evaluation of the significance of a murmur, particularly of one heard over the cardiac apex, the decision rests almost entirely with factors other than those relating to the murmur itself. This view has been stressed by Mackenzie,[1] Seham,[4] Gibbes [19] and others. The murmur heard early in the course of an acute rheumatic infection is frequency soft and blowing, and, per se, may be indistinguishable from a functional murmur. However, the history together with symptoms and signs of the disease should serve to differentiate the condition. An electrocardiogram may be of aid. Functional murmurs are, as a rule, distinguishable from those of chronic valvular disease by the absence of rheumatism and cardiac symptoms and of cardiac enlargement or other objective signs of cardiac disease. In doubtful cases roentgen ray examination repeated, if necessary, in the form of orthodiagrams may prove of great assistance. It is frequently inadvisable and impossible to decide definitely the significance of a murmur by a single examination. Observation over a prolonged period of time may be necessary. Work like that of Lincoln,[18] whose observation of children over a period of years proved all murmurs encountered to be nonpathologic, is valuable. Although Feinberg and Steuer [25] included cases of gross cardiac hypertrophy in their "uncomplicated group," their finding that in the presence of a normal orthodiagram serious complications are unlikely to develop in a patient with a systolic murmur is interesting.

Tests for cardiac efficiency are of limited value in determining the significance of murmurs. From the work of Barringer [26] on adults and from that of Wilson [27] on children it is obvious that the "exercise tolerance" test by which the reaction of the pulse to exercise is measured is unreliable as a test of cardiac function. In almost all instances among the boys under consideration the influence of exercise on the functional murmur itself was one of accentuation.

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24. Chapin, H. D., and Royster, L. T.: Diseases of Children, ed. 6, New York, William Wood & Company, 1928.

25. Feinberg, M. H., and Steuer, L. G.: Apical Systolic Murmurs in Children, Follow-Up Observation in One Hundred Cases, Am. Heart J. **7**:553 (June) 1932.

26. Barringer, T. B.: Studies of the Heart's Functional Capacity, Arch. Int. Med. **20**:829 (Dec.) 1917.

27. Wilson, M. G.: The Circulatory Reactions to Graduated Exercise in Normal Children, Am. J. Dis. Child. **20**:188 (Sept.) 1920.

MURMURS PRODUCED BY EXERCISE

While reference to murmurs produced by exercise is occasionally made in the literature, apparently few systematic studies regarding this phenomenon have been carried out. Norris and Landis [28] mentioned the study of Munford, who found that in 7.48 per cent of students heart murmurs developed after the exercise of chinning themselves as often as possible, and that of Mackenzie who found an incidence of 27.8 per cent of murmurs following exercise in apparently healthy students.

Table 1 shows a surprisingly high incidence of murmurs following exercise. The incidence might have been even higher had the exercise (which consisted of hopping for about one-half minute) been more strenuous. A comparison of the results recorded in table 1 with those

TABLE 2.—*Incidence of Venous Hum at Different Ages*

Age	Number of Cases	Venous Hum	
		Number	Per Cent
5	11	8	73
6	17	16	94
7	15	13	87
8	20	15	75
9	17	9	53
10	18	12	67
11	16	5	31
12	15	11	73
13	19	11	58
14	27	8	30
15	34	8	24
16	33	8	24
17	22	6	27
18	11	4	36
Total	275	134	49

on a group of 54 track athletes tends to confirm the finding of Mackenzie [29] that murmurs following exercise are slightly predominant in nonathletic persons. A lack of "training" may also explain the much higher incidence in the younger age groups. According to Norris and Landis,[28] murmurs heard following exercise are "generally ascribed to loss of muscle tone affecting chiefly the mitral sphincter." In contradistinction to murmurs before exercise, murmurs following exercise are more commonly heard over the apex alone than over the base alone. Most commonly they are heard over both areas.

While the murmurs are transitory and evidently inconsequential, they may be of considerable indirect significance because they are probably attributable to a more energetic action of the heart. Factors other than exercise resulting in greater cardiac activity may also produce murmurs. Thus Hochsinger [17] has pointed out that murmurs present

28. Norris, G. W., and Landis, H. R. M.: Diseases of the Chest and the Principles of Physical Diagnosis, ed. 3, Philadelphia, W. B. Saunders Company, 1924.

29. Mackenzie, R. T., cited by Norris and Landis.[28]

at the beginning of an examination (at a time of psychic stimulation) may disappear before its termination. The murmurs frequently heard during acute fevers may probably also be ascribed to accelerated cardiac action.

THE VENOUS HUM

While the venous hum is an extracardial phenomenon, it may, nevertheless, be discussed with propriety and advantage in connection with "functional" cardiac phenomena because of its occasional audibility over the precordium and because of its nonpathologic nature. The term "venous hum" coined (according to Landis and Kauffman [30]) by Hope as early as 1839 denotes a murmur at times low-pitched and rumbling but usually humming, heard over the internal jugular veins at the base of the neck (sternoclavicular junctions and inner thirds of the clavicles). The sound is continuous with rhythmic (diastolic) accentuation. Its intensity varies greatly. As pointed out by Norris and Landis,[28] many explanations as to the genesis of the hum have been offered. The preponderance of right-sided hums favors the view that the maintenance of an almost constant caliber of the right jugular vein (which is practically a direct continuation of the right innominate vein) by the cervical fascia and the consequent incomplete adjustment to the rate of blood flow result in a murmur. On the other hand, the production or accentuation of a hum by turning the head up and away from the auscultated side strengthens the view that it is due to compression of the vein against the transverse processes of the cervical vertebrae. The fact that the results recorded in table 2 were obtained with the subject looking straight forward may explain the somewhat lower incidence than that reported by Landis and Kauffman.[30]

While the venous hum is of no pathologic significance, errors in its interpretation may lead to disastrous results. It is conceivable that a venous hum might be mistaken for a sign of aneurysm and the mistaken diagnosis might lead to unnecessary operation. Furthermore, a venous hum may prove perplexing because of its audibility over the precordium. Possibly many instances of so-called Eustace Smith's signs are nothing more than venous hums.

SUMMARY

On the basis of evidence from the literature and personal observation an attempt has been made to point out the insignificance of presumably nonorganic auscultatory cardiac findings commonly encountered in children. While the inconsequential nature of the phenomena has been stressed, emphasis on due care in their proper evaluation has not been omitted.

30. Landis, H. R. M., and Kauffman, I.: The Occurrence of Venous Hums in Children, Arch. Pediat. **29**:88 (Feb.) 1912.

Pediatric Biographies

WILLIAM DOUGLASS

1691-1752

NOTES ON THE HISTORY OF SCARLET FEVER

JOHN RUHRÄH, M.D.
BALTIMORE

In the early part of the eighteenth century there came to America a group of Scotch physicians, among them Cadwallader Colden of New York, Lining, the elder, Moultrie, Chalmers and Garden of South Carolina. Another, a very remarkable man, William Douglass, settled in Boston.

Sydenham had been dead three years when Douglass was born, in Gifford, County of Haddington, Scotland, in 1691. He is said to have received the degree of master of arts at Edinburgh and of doctor of medicine at Utrecht. He also studied at Leyden, and knew Greek, Latin, French and Dutch. In 1716 he went to Boston, and, after a year of travel in the West Indies, he returned to that city in 1718, where he practiced medicine the rest of his life. In 1721, when Zabdiel Boylston began inoculating for smallpox, Douglass wrote, "The Abuse and Scandals of Some Late Pamphlets in Favour of the Small-Pox," which appeared in 1722. He had previously treated with contempt Cotton Mather's communication regarding Timoni's work in Constantinople. He later recognized that he was in the wrong, and, strong man that he was, wrote, "A Practical Essay Concerning the Small-Pox" and "A Dissertation Concerning Inoculation of the Small-Pox."

Douglass, like Waterhouse, was not popular with his fellow practitioners, for he was fearless and expressed his opinion freely. He was one of the men who founded a medical society, and he favored the regulation of the practice of medicine and an examining board. He was a bachelor, and died in Boston on Oct. 21, 1752.

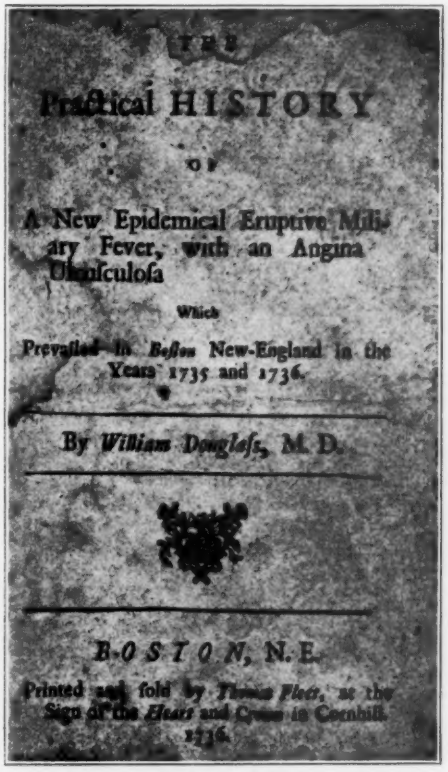
He also wrote "A Summary, Historical and Political, of the First Planting, Progressive Improvements and Present State of the British Settlements in North America." This consisted of two volumes and was printed in Boston in 1749 to 1751 and in 1755, and in London in 1760. The book[1] was reprinted in 1897 with an excellent biographic sketch by Charles J. Bullock. Douglass also published open letters to Thomson, Smellie and Bolylston.

His chief contribution, an American pediatric classic, is "The Practical History of a New Epidemical Eruptive Miliary Fever; with an

1. Economic Studies, New York **2**:265, 1897.

Angina Ulcusculosa Which Prevailed in Boston in the years 1735 and 1736." This was published in Boston, by Thomas Fleet, in 1736, and was printed by request in the *New England Journal of Medicine and Surgery.*[2]

Morris Casper reprinted it in 1758 in the second edition of his "Essay on the Pathology and Therapeutics of Scarlet Fever," and George H. Weaver reproduced it, much reduced in size, as an illustration in his


THE
Practical HISTORY
OF
A New Epidemical Eruptive Military Fever, with an Angina Ulcuſculoſa
Which
Prevailed in *Boſton* New-England in the Years 1735 and 1736.
By *William Douglaſs*, M. D.
BOSTON, N. E.
Printed and ſold by *Thomas Fleet*, at the Sign of the *Heart* and Crown in Cornhill.
1736.

Title page from Douglass' book.

most excellent account of Douglass in the *Bulletin of the Society for Medical History of Chicago,*[3] which should be consulted by those interested.

Douglass' contribution, about twelve pages in length, is a superb piece of clinical description. Like most physicians, he was not good at theorizing; his speculations are of little value, but as one reads his opinions and his reasons for them, one is reminded of the speculations

2. New England J. Med. & Surg. **14**:1, 1825.

3. Bull. Soc. M. Hist. Chicago **2**:229, 1917-1921.

in present-day literature on the epidemiology of poliomyelitis, which in the language of Bunge, are made up chiefly of quotations and errors.

There are wide differences of opinions on the part of medical historians as to the nature of the New England epidemic. Hirsch, Creighton and Weaver believe that it was scarlet fever, and Haeser, Green and Packard, that it was diphtheria. This variation has been explained by a careful and thorough medical historian, Ernest Caulfield, who, after a long and exhaustive study of the New England throat distemper, is of the opinion that in Boston the epidemic was scarlet fever and in the other places, diphtheria. Apart from the description of Douglass, the fatality rates differ greatly, that of the Boston epidemic of from 1.7 to 2.8 per cent comparing with the 2.9 per cent of scarlet fever in Connecticut (1895 to 1929), and the New Hampshire fatality rate of from 16 to 60 per cent (1735 to 1736) comparing with the 26 to 49 per cent of the preantitoxin days in New York and Boston (1880 to 1887).

THE PRACTICAL HISTORY OF A NEW EPIDEMICAL ERUPTIVE MILIARY FEVER, WITH AN ANGINA ULCUSCULOSA, WHICH PREVAILED IN NEW-ENGLAND IN THE YEARS 1735 AND 1736

THIS Distemper did emerge 20th *May*, 1735 in *Kingston* Township 50 Miles Eastward from *Boston;* it was no foreign importation, *Kingston* being an inland place, of no Trade or considerable communication. The first seized was a *child who died in three Days Illness;* about a Week thereafter in another Family at four Miles distance, three children were seized successively, and died also the third Day; it continues spreading gradually in that Township, seizing here and there particular Families with that degree of violence, that *of the first circiter forty decumbents none recovered* as we were informed. It was vulgarly called the *Throat Illness,* or *a Plague in the Throat,* and alarmed the Provinces of *New-England* very much. Some died of a sudden or acute *Necrosis;* but most of them by a *Symptomatick affection of the Fauces or Neck;* that is by *Sphacelations* or corrosive *Ulcerations* in the *Fauces,* or by an *infiltration* and *tumefaction* in the Chops and fore part of the Neck, so turged, as to bring all upon a level between the *chin* and *sternum,* occasioning a *strangulation* of the Patient in a very short time.

After a few Weeks it spreads into the neighbouring Townships, but with more mildness. The first *appearance* that we can recollect of it in *Boston,* was 20th *August* on a Child of Capt. *Stannys* at the North End; having *white specks* in the Throat, and a *cutaneous efflorescence:* A few more in the same Neighbourhood were seized in like manner, about the same time. Towards the end of *September* it appeared in several parts of the Town, with a complaint of *soreness in the Throat, Tonsils swelled and speckt, Uvula relaxed, slight Fever, flush in the Face, and an Erysipelas like efflorescence on the neck, chest* and *extremitise;* but being of no fatal or bad consequence, nothing more than a common cold was suspected. Our first alarm was from a young man *How aet. 20* in the beginning of *October:* His *History* runs thus; He was lately arrived from *Exeter* to the Eastward, where his Brother died of this Illness; his *Symptoms* were great prostration of Strength, a speck in one of the *Tonsils,* colliquative *Sweats, Pulse* not high and full, but low, hard, stringy, unequal and more frequent than natural, *deglutition* good to the last, no *Sphacelation* in the Throat, no eruption; from a rash inconsiderate

opinion of forcibly quelling the *Malignity,* he was thrice let Blood, had some Emeticks and Catharticks administered, and *by profuse evacuations was gradually reduced, so as to die of a gentle decay of natural Strength,* the 6th day of Illness.

Beginning of *November* it spread considerable in *Boston,* especially amongst *Children,* with more violent Symptoms, and severals die of it in various Periods: it seemed to be at the hight, as to Numbers ailing and quantity of Deaths, the second Week of *March;* that Week there were 24 Burials, whereas *communibus annis* in that Season they are only 9 or 10 per Week.

It is generally in so considerable a degree *more favourable in Boston,* than in the Townships where it first prevailed; that many can scarce be persuaded of its being the same Distemper: It is nevertheless essentially the same, there is no Symptom, even the most malignant that has appeared in *New Hampshire,* but what the like has occurred in *Boston.* . . .

. . . A previous *listlessness,* and languishing countenance for a Day or two, or some other *proenuncia* as u. g. wet Nurses loosing their Milk. The first attack is somewhat of a *chill* or shivering; soon after follows *head ake* or some other versatile *spasmodick pains,* as pain in the back, joints, side, &c; a vomiting or *nausea,* or in some constitutions which are not easily provoked to vomit, only a certain uneasiness or sickness at the Stomach; at the same time the *Uvula* but chiefly the *Tonsils* were tumified, inflamed and painful, with some white *specks;* then follows a flush in the Face and some *miliary eruptions* there, with a benign *mild Fever,* the same efflorescence soon after appears on the neck, chest and extremities; the 3d or 4th Day, Eruption is at the hight and well defined with fair intervals; the flushing goes off gradually, with a general *itching;* and in a day or two more the *cuticle* scales or peels off, especially in the extremities: At the same time the cream coloured sloughs or specks in the *Fauces* become loose and cast off, and tumefactions there do subside. The tongue from the beginning is fur'd as in a *Mercurial ptyalism,* urine high coloured, Blood by U. S. more *florid* than natural, in the whole course of the distemper a very *great prostration of Strength,* and faintness upon recovery, *nervous pains* and weakness in the joints, particularly in the neck, wrists and ancles; universal tenderness to the touch; a tickling guttural cough, some short *Hectick flushings,* and loss of *en bon point.* As in the Measles there is a peculiar smell, so in our Distemper the *effluvia* from the Patient have a proper smell; in Children as if troubled with *Worms,* in grown Persons the *rancid smell* of foul Bed Linnen. The *alvine excrement* is of a dark cast and very fetid. . . .

For a more distinct conception of the *varieties in this Distemper,* they may be reduced to three *classes.*

I. Those who die the first, second and third day of Illness, by an irremediable *Necrosis of the Oeconomy:* in such the Seizure is generally sudden, a sinking pain at the Stomach, an extreme prostration of Strength, a titubating low pulse, in some a stupor, in others a delirium, in some children convulsions, and all of them generally die dozie: they are attended with some *colliquation,* as continued vomiting, purging, profuse Sweats, bloatedness of the habit, an infiltration like that of the *Mumps* vulgarly so called, one or more of these: in general the texture of their blood and juices is much destroy'd and rendred an incoherent puddle of corruption; in fact immediately upon (sometimes before) their *exit,* they have an intolerable *foetor.* In this Class U. S. and other evacuations did accelerate death.

II. Those where the distemper has its common or ordinary course; here the 6 or 7 day seems to be *critical,* and the Symptoms of death or recovery do generally then begin to manifest themselves. Some by peculiarity of constitution, and

from improper administrations do die or have an incipicent recovery sooner: others for the like reasons or some particular accidents (u. g. if about the time of regular *menstruation,* the complicated fret occasions worse Symptoms, and of longer continuance) have this period protracted and in such (where death is inevitable) the Symptoms of death may continue a day or two longer, that is the Patient may die the eighth or ninth day. All who continue Ill after that period, belong to the third Class, that is of consequential ails.

The Symptoms of *bad Omen* in this Class, are very great prostration of strength, dejection and despondency of mind, titubating low pulse, incessant vomitings purgings or sweats, Tonsils much inflamed endangering strangulation, the specks in the *Fauces* of a brownish or leaden colour, or ragged and jagged, a continued *jactantia* in some, in others a *stupor,* refusal of *assumenda* even of diluting common drinks, a dry parched skin, Eruptions appearing and disappearing alternately, Eruption universal of a dark redish cast continuing *crude* many days (because in this as in all eruptive Fevers, the darker or more livid the efflorescence, the more malignant) where the *miliary* pustles are large, distinct and pale like a *chrystaline Small-Pox;* where strong Cordials and *Alexipharmicks* have been used, the face, eye-lids, arms, hands, legs, feet swell, and are of a dark red complexion, as in the most malignant Small Pox; in children if the *velum Palati* be much affected, with an ichorous discharge by the Nose; where many *mucous* linings are expectorated, resembling the cuticle raised by Vesications; when *pus* was brought up, where no sloughs or exulcerations could be seen in the *Fauces;* where without any difficulty in swallowing, this affection has reached down the *Bronchia* unto the Lungs with the Symptoms of a *New-England Quinsey,* and was erroniously deemed such: the deeper in the *Thorax* the complaint the greater the danger: in some young children with scarce any appearance in the Throat, spreading Ulcers did form behind the Ears in the place where Infants have a natural Issue or running. In some the Tongue did throw off a slough or *Exuvia,* retaining the impressions of the Papillae; being a *Mucus* inspissated, and of the same nature with those mucous linings expectorated from the *Bronchia* or *Oesophagus.* Some have had impostumations in the Fauces, with a fatal strangulation, while others have escaped by the discharge of *Ichorous* curdly matter. Some especially of the adult female kind, have had *Hysterical* or Nervous Suffocations; but of no bad consequence, unless officiously and ignorantly treated with U. S. and other evacuations.

SCHOLIA *or some general remarks upon the whole.*

1. *This seems to be a new kind of Epidemical disease.* It is not the same with the *Aphthae* which have at times prevailed in *Holland,* as described by *Forestus,* and mentioned by *Boerhaave* in his Colleges. Tournefort says there is a distemper not uncommon in the *Levant, viz* a Carbuncle or plague sore in the bottom of the Throat; it carries off children in a few days, but does not affect grown People as does ours. Capt. *Morton* of late *Plymouth* Colony, who wrote many years ago his *New England Memorial,* says that an. 1650. a disease in the Mouth and Throat prevailed, which proved mortal to many in a short time; but he does not describe it, and mentions nothing of a Fever. In *Boston November* 1719. a slight miliary fever chiefly with children, but was over in two or three days, unless by catching cold it continued appearing and disappearing alternately for some days longer; there was no complaint of the Throat, and no deaths ensued. It is not the same with the sore Throats which are observed from time to time in some of our Country Towns, especially in the Winter season: these are Endemial and constitutional, being tumefactious and exulceratious with fluxion in the *Fauces* and *Neck;*

proceeding from an intense scrophulous, scorbutick, or *Psorick* habit (in such subjects vesications by *Cantharides* did putrifie) without any Eruptive fever: ours have generally an Eruptive fever or tendency that way, so that of those who have died in *Boston,* not above one in seven died of any Throat ail, but of this fever. It is however observable that the Scrophulous and Psorick, are most susceptible of this distemper, and suffer more remarkably.

2. *This Epidemical distemper is no creature of the Seasons,* it having prevailed from *May* 1735, when it first emerged, the whole year or all the Seasons round. *It is no produce of peculiar climates and soils,* because it hath made its appearance in various places from *Pemaquid* in 44 N. Lat. to *Carolina* Southward, and as we are lately informed, it is in our *West India Islands.* It is remarkable that in damp places, as near large Ponds, fresh water Rivers, woodlands, and the like, it has done the greatest execution, as does the *Rot* amongst *Sheep* in fenny lands. . . . We have *Anatomically* inspected Persons who died of it with so intense a *foetor* from the violence of the disease, that some Practitioners could not continue in the room; but without being infected our selves or carrying it into families. Many children without reserve, frequent the houses and chambers of the sick, and escape. *It does therefore proceed from some undiscovered quality of the air, affecting only peculiar constitutions of persons and families:* notwithstanding of its being generally favourable, it proves fatal to certain families; many families for this reason have buried all or most of their children, u. g. *Boynton* of *Newbury-Falls* lost his eight children, at *Hampton-Falls* in 5 families died 27 Persons.

4. In some who were very slightly affected, their Illness was of a much shorter continuance, than is described in the Standard kind. *Most of those who died of the Physician died by immoderate evacuations.* As to the deaths, only a few were occasioned immediately by any distemperature of the Throat; they were generally the effect of the Fever, either by an immediate *Necrosis* at first seizure, or by the ordinary fatality of Fevers, or by consequential ails. In *Boston* at a medium of the last eight healthy years (1723, 1724, 1725, 1726, 1727, 1728, 1732. and 1733) in the Months of *October, November, December, January, February, March, April* to 18*th May,* died pr. an. 263 Whites and Slaves; this year in the same space of time died 382, is 114 *extra* deaths, and may be reasonably charged to this Illness, it being otherways a healthy time: of these 114, about 71 cases came to my knowledge, whereof in the first period died 35, in the second period 28, and of consequential ails, 8. Of these 71, only about 10 can be said to have died of sore Throats. Of these 71 only 9 were upwards of 14 aet. According to the nearest estimate I can make in round numbers, about 1 in 35 have died, that is about 4000 Persons in *Boston* have had this distemper, which is about one 4th part of the Inhabitants.

5. The Summer 1735 was unusually wet and chilly with many Easterly winds, in the Summer & Autumn it prevail'd and was very mortal in several Country Towns. In Boston it began in Autumn but did not prevail until Winter, which was not rigid with hard frosts as is usual, but with a very disagreeable chill in the air, especially in the Month of *March last,* in which Month was our greatest Mortality.

Pediatrics in Art

ABDOMINAL BINDERS

JOHN RUHRÄH, M.D.
BALTIMORE

A good example of one of the many styles in abdominal binders is that seen in a painting after the manner of Lorenzo Lotto which hangs in the Dresden Gallery. Here is seen a beautiful Madonna with three

A painting from the Dresden Gallery by V.-A. Bruckmann, showing one type of abdominal binder.

saints, one of whom may be St. Francis of Assizi, judging from the stigmata of the crucifixion. The chubby infant on its mother's arm is attentively examining the scroll held by one of the other saints. The story of the abdominal binder, an extremely interesting one by the way, could almost be completely told by reproduction of works of art.

News and Comment

GENERAL

Archives of Pediatrics, Anniversary Number.—The February issue of the *Archives of Pediatrics* commemorates the fiftieth anniversary of this journal. The original number, the first in this country to devote itself exclusively to the diseases of infants and children, appeared on Jan. 15, 1884. The February issue is dedicated to the pediatric pioneers of 1884 and contains reprints of articles contributed to the *Archives* during the course of its first year by distinguished clinicians of that time, including Drs. William T. Plant (Convulsions in Children), A. Jacobi (Congenital Lipoma), J. Lewis Smith (Hygienic Management of the Summer Diarrhea of Infants), John M. Keating (Infant Feeding), F. Forchheimer (Rachitis), Louis Starr (Intestinal Indigestion), L. Emmett Holt (Valvular Disease of the Heart: Purpura Haemorrhagica; Malarial Fever; Sub-Acute Rheumatism, Complicated by Pericarditis with Effusion, and This by Pneumonia—All Occurring within a Period of Eight Months, with Ultimate Recovery), William P. Northrup (Expiratory Dyspnoea from Enlarged Bronchial Glands in a Case of General Tuberculosis and Empyema) and Henry Dwight Chapin (A Case of Pseudomembranous Colitis).

Course in Child Development, University of Iowa.—The State University of Iowa will carry on its usual summer session in child development and parent education from June 11 to August 23. The tuition fee is $36 for the first summer and $30 for the second.

UNIVERSITY NEWS

Dr. Wilhelm Bayer, of the University Children's Clinic of Hamburg, has been named medical director of the Hamburg Infants Home, replacing Prof. Dr. Bauer.

Dr. Walter Keller, of Heidelberg University, has been named associate professor and chosen as the director of the Pediatric Clinic of the State Hospital at Mainz.

Dr. Grävinghoff has been named associate professor and director in the Pediatric Clinic of the University of Münster.

PERSONAL

Dr. Armand-Delille of Paris has been promoted to Officer of the Legion of Honor.

DEATHS

Adolphe Pinard, director and founder of the School of Child Hygiene of the Faculty of Paris, died recently at the age of 90.

Dr. Paul Grosser, professor of pediatrics at the University of Frankfort-on-Main, died recently at the age of 54.

Abstracts from Current Literature

Anatomy and Physiology

ESTIMATION OF CARDIAC AREA IN CHILDREN. P. C. HODGES, W. ADAMS and W. GORDEN, J. A. M. A. **101**:914 (Sept. 16) 1933.

There has been a need of tables and formulas for predicting the normal size of the heart of a child from height and weight. The authors' teleoroentgenographic studies of 169 carefully selected normal children of varying ages form the basis for such an equation. A table is published which permits rapid estimation of the normal size of the heart without computation. BONAR, Salt Lake City.

UNUSUAL APPEARANCE OF THE SELLA TURCICA. RABAT LEROUDIER, Bull. et mém. Soc. de radiol. méd. de France **20**:154 (March) 1932.

A girl of 12 years presented marked retardation in growth and had suffered for three years from progressive impairment of vision. Roentgen examination of the pituitary region showed a considerably enlarged sella turcica. Roentgen treatment was instituted, resulting in great diminution of the visual difficulty and in increased growth. Three years later the visual improvement continued, but the girl, in spite of continuing to grow, showed none of the secondary sex characteristics—development of the breasts, pilosity or menstruation. Infantilism, therefore, was present, and not merely pituitary nanism.

A fresh roentgenogram showed the sella turcica enlarged and two-thirds filled with a tissue so dense as to appear as a thick cushion of neoplastic material covering the whole surface of the sella. The author is inclined to regard this neoplastic tissue as the result of a proliferative osseous process induced by diminution of volume in the pituitary tumor under the influence of roentgen therapy, constituting an indirect indication of the progress of the tumor toward cure.

VINTON, Brooklyn.

LYMPHATIC VESSELS OF THE GREAT OMENTUM. H. ZSCHAU, Deutsche Ztschr. f. Chir. **240**:27 (May 22) 1933.

Zschau briefly reviews the literature regarding the disputed question as to whether or not lymphatic vessels are present in the great omentum. His procedure consisted in removing this structure from a freshly killed dog, keeping it moist by placing it on a towel saturated with Ringer's solution and injecting india ink into the fatty structures. By this simple method the india ink was observed to be taken up in numerous lymphatic vessels in the fat of the great omentum. The lymphatic vessels could be easily traced to the attachment of the great omentum to the greater curvature of the stomach, and were here observed to join the lymphatic system of the stomach. GLASER, Rochester, N. Y.

Pathology, Bacteriology and Biochemistry

DIFFERENTIATION AS EFFECTED BY DIIODO-TYROSINE PLUS CERTAIN OTHER AMINO ACIDS. OLIVE HOFFMAN and F. GUDERSNATSCH, Endocrinology **17**:239 (May-June) 1933.

Tadpoles were kept on a diet in which amino-acids added to the water formed the sole source of nitrogen. The effect of different acids and combinations of acids was noted as regards their ability to maintain the animals, to cause growth and to cause differentiation, e. g., the appearance of legs, the beginning of absorption

of the tail and other signs of development. Some alpha amino-acids—phenylalamine, tyrosine and tryptophan, in the order of increasing effectiveness—exert on differentiation an accelerating influence which is comparable in quality to that of diiodo-tyrosine (thyroxine), though it is far less pronounced in quantitative effect. When the two iodine molecules in diiodo-tyrosine are increased to four to produce thyroxine, the actual hormone, the intensity of the effect of differentiation increases still further. It is suggested that because of the definite structural similarity, these amino-acids are precursors of the thyroid hormone and carry a linkage which makes for differentiation. It also becomes clear why iodized proteins accelerate differentiation. Another group of amino-acids—arginine, lysine and cystine—facilitates growth, but no chemical relationship between these and the "growth" hormones has been established.

JACOBSEN, Buffalo.

APPARATUS FOR THE RAPID STUDY OF ULTRA-VIOLET ABSORPTION SPECTRA. J. S. PHILPOT and E. H. J. SCHUSTER, Medical Research Council, Special Report, Series no. 77, London, His Majesty's Stationery Office, 1933.

This report describes new methods for physical investigation of absorption spectra that have been devised by members of the staff of the Medical Research Council at the National Institute for Medical Research, Hampstead. The apparatus is especially recommended for measurements of the ultraviolet absorption spectra, in which rapid operation is as important as accuracy in the case of unstable mixtures present in the formation of vitamin D. Five seconds is sufficient to provide a complete absorption curve.

The principle of the apparatus is based on a photographic photo-electric method. It provides measurements at all wavelengths from one spectrogram of the solution and one of the solvent. The method involves the use of a wedge spectrogram produced by means of a logarithmic stop and a cylindric lens so that the aperture decreases exponentially from the top to the bottom of the spectrogram. This is combined with the split beam principle, in which part of the light passes through the cell containing the absorbing liquid while part does not. Points of equal intensity are determined by the photo-electric densitometer, the instrument of Dobson being modified so that such points are found in one operation by varying the distance apart of two spots of light. Speed has also been gained by means of a machine for working out results and plotting them on curves (the photometrograph) and by a machine for analyzing the curves obtained (the mixigraph). The whole process of measuring, working out and plotting can be done at the rate of two of these points a minute.

The authors claim that the method is fairly satisfactory as regards error because (1) it does not involve the "Schwarzschild factor"; (2) other forms of systematic error are readily detected if present; (3) it does not require a constant light source or photographic plates of constant sensitivity except at points near together on the same plate, and (4) the random error has a standard deviation of about 1 per cent when the extinction coefficient of 1 is measured.

SCHLUTZ, Chicago.

THE MINIMAL LETHAL DOSE OF SODIUM TAUROCHOLATE GIVEN INTRAPLEURALLY. P. SCHIAPARELLI, Riv. di clin. pediat. **31**:257 (March) 1933.

In healthy rabbits, the minimal lethal dose of sodium taurocholate injected intrapleurally is 0.12 Gm. per kilogram of body weight. The degree of dilution in salt solution or the administering of the drug in blood serum did not affect the lethal dose. The lethal dose when given intravenously is 0.5 Gm. per kilogram.

If death occurs within a short period after the injection, little or no change in the lung is found at autopsy. If death occurs after a longer time, as thirty days, complete hepatization (carnification) of the lung is found.

The healthy pleura is probably more sensitive to the drug than is an infected pleura (empyema). The fibrinous and fibrous coating delays the absorption; thus a triple advantage exists in its therapeutic use: (1) It is more slowly absorbed, (2) it does not involve the lung tissue, and (3) it remains longer in the pleural cavity to act as a bactericidal and a bacteriolytic agent.

HIGGINS, Boston.

CHANGES PRODUCED IN THE OSSEOUS LABYRINTH OF GUINEA-PIGS BY OVERDOSAGE OF VIOSTEROL. A. TOBECK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:550 (March 20) 1933.

Tobeck reports that by overdosage of viosterol he was able to produce changes in the osseous labyrinth of guinea-pigs. These changes consisted, in the main, in the appearance of a marrow abundant in cells and containing giant cells, in an osseous decomposition through lacunar resorption and perforating canals and in new osseous growth through the development of osseous trabeculae and margins. It may be assumed that the viosterol caused a disturbance of the calcium metabolism that resulted in the severe osseous changes. In view of the fact that the results of the latest investigations place otosclerosis among the large group of osteodystrophic bone diseases, the production of osseous changes in the otic capsule resembling otosclerosis by overdosage of viosterol is significant.

EDITOR'S ABSTRACT [ARCH. OTOLARYNG.]

MICRODETERMINATION OF IODINE IN THE BLOOD, BODY FLUIDS AND ORGANIC MATERIALS. J. EYCKERMAN, Ztschr. f. Kinderh. **54**:435, 1933.

The method consists in the rapid ashing of the organic material with hot sulphuric acid and a proprietary 30 per cent solution of hydrogen dioxide, the reduction of the iodine with sodium sulphate and the distillation and the titration of the iodine. The original paper is to be consulted for details.

SHELLING, Baltimore.

Nutrition

THE EFFECT OF THE PASTEURIZATION OF MILK ON THE UTILIZATION OF ITS CALCIUM FOR GROWTH IN THE RAT. MARTELL ELLIS and H. H. MITCHELL, Am. J. Physiol. **104**:1 (April) 1933.

The pasteurization of milk slightly lowers the availability of its calcium for the growing rat, possibly by destroying some unknown constituent of the milk that favors its utilization in the body.

NOURSE, Cleveland.

THE IMPORTANCE OF INFANT FEEDING TO THE GENERAL PRACTITIONER. RALPH R. SCOBEY, Arch. Pediat. **50**:110 (Feb.) 1933.

The important rôle of the general practitioner in any program of child health is pointed out. The neglect of the dietetic management of infants is stressed, and a method of infant feeding is presented which the author believes to have certain elements of simplicity as compared with the methods in general use.

WADDELL, JR., University, Va.

RECENT CLINICAL EXPERIENCES WITH 3,800 INFANTS ON EVAPORATED, POWDERED AND CONDENSED MILKS: A REVIEW. JAMES A. TOBEY, Arch. Pediat. **50**:183 (March) 1933.

Twenty-five clinical investigations on the use of evaporated, powdered and condensed milk in the feeding of infants are summarized. Tobey thinks that a review of the various investigations reveals that these processed milks not only have no disadvantages but possess certain distinct qualities which justify their

present vogue; no adverse findings have been reported. Among the principal advantages suggested for the concentrated milks are their exceptional digestibility, uniformity of composition, cleanliness and bacterial safety, high nutritive values, easy assimilability of calcium and phosphorus, convenience for making concentrated formulas, general availability, keeping qualities, simplicity of use and economy.

WADDELL, JR., University, Va.

VEGETABLE MILK IN INFANT FEEDING. J. KLEIN, Arch. Pediat. **50**:205 (March) 1933.

Two cases of seborrheic eczema are reported healed on a diet of soy bean flour. Attention is called to the composition, qualities and indications for the use of vegetable milk in the feeding of infants, and it is suggested that cocoanut milk has a higher biologic value than any other vegetable protein.

WADDELL, JR., University, Va.

SPECIFIC HYPERSENSITIVENESS TO OWN BREAST MILK. W. W. DUKE, J. A. M. A. **98**:1145 (April 23) 1932.

A woman with a strongly allergic family history gave a history of prolonged eczema. She was sensitive to egg and heat and to injections of her own breast milk, a few drops of which continued to flow for seven years after the weaning of her last child. She lost the sensitivity by subcutaneous injections of her breast milk, an egg-free diet and treatment for increased tolerance to heat. The egg-free diet and the treatment for intolerance to heat were tried first, but did not give complete relief. The subcutaneous injections of the breast milk also stopped the secretion of the breast milk.

HOYER, Cincinnati.

VITAMINS IN PRACTICAL EXPERIENCE. LESLIE J. HARRIS, Brit. M. J. **2**:231 (Aug. 5) 1933.

The author expresses his views on the requirements of vitamins and on vitamin therapy.

ROYSTER, University, Va.

HYPOGLYCAEMIA: A SURVEY AND AN ACCOUNT OF TWENTY-FIVE CASES. C. SIPPE and J. BOSTOCK, M. J. Australia **1**:207 (Feb. 18) 1933.

The authors report the results of a study of twenty-five cases of hypoglycemia with clinical manifestations, twenty-four of which occurred in females. They conclude that a blood sugar content of 0.09 Gm. or less per hundred cubic centimeters during fasting, in the presence of typical symptoms, is diagnostic. In a large series of medical cases the percentage of cases of hypoglycemia was 0.47, and the percentage of cases of diabetes, 0.51.

Hyperinsulinism due to nerve or endocrine dysfunction or to new growths of the pancreas, disturbances of carbohydrate metabolism, damage to the liver, muscular dystrophies, functional disturbances of the glands of the endocrine system other than the pancreas, a lowered renal threshold in disease of the kidneys and allergy are given as the etiologic factors in the production of the syndrome of hypoglycemia.

The important symptoms are noted, and the nervous and psychic phenomena are stressed. The syndrome is classified in two main groups, the true and the relative. The first group is subdivided into the acute fulminating, the chronic and the symptomless types. The second group is subdivided into two groups, which include the cyclic vomiting of childhood and the simple types in which the normal amount of blood sugar is insufficient for the requirements of the person. Treatment consists in the administration of adequate amounts of dextrose and the removal of the etiologic factors.

GONCE, Madison, Wis.

DIGESTION OF STARCH IN INFANTS. M. BESPALOFF, Nourrisson **20**:352 (Nov.) 1932.

In order to determine the extent of the digestion of starch in infants, smears of stools were made and stained with a dilute solution of iodine. These were then examined microscopically, the undigested starch giving the usual blue reaction. To render starch suitable for digestion by infants under 1 year of age, it should be thoroughly cooked for from two to three hours in a double boiler or from thirty to forty-five minutes directly over the flame.

Of healthy infants under 6 months of age, 30 per cent are able to digest starch without trouble, and 50 per cent adapt themselves to it after short periods. Practically all infants over 6 months of age are capable of digesting starch without difficulty, whether they are sick or well.

The introduction of starches in the diet of breast-fed infants under the age of 6 months is not advisable. Two positive indications for the early introduction of starchy foods in the case of artificially fed infants are: (1) pylorospasm and (2) dyspepsia due to faulty digestion of cow's milk, when it is believed that the fermentation caused by the digestion of starch will be helpful in combating the putrefaction of casein.

McCLELLAND, Cleveland.

THE USE OF EVAPORATED MILK IN INFANT FEEDING. G. BLECHMANN, Nourrisson **21**:101 (March) 1933.

Attention is called to the fact that babies fed on evaporated milk require antiscorbutic fruit juices from early infancy. The author feels that for the requirements of infant feeding there should be three kinds of evaporated milk: (1) that prepared from skimmed milk, (2) that prepared from half-skimmed milk and (3) that prepared from whole milk. The exact composition of each should be clearly indicated on the label.

McCLELLAND, Cleveland.

CONCENTRATION OF LOWER FATTY ACIDS IN HUMAN MILK SECRETED UNDER NORMAL CONDITIONS AND DURING THE MENSTRUAL PERIOD. FERNANDA BORSARELLI, Riv. di clin. pediat. **31**:664 (June) 1933.

Twelve specimens of human milk secreted during the menstrual period and thirty-eight specimens secreted during a normal period were analyzed and compared as to the content of fatty acids of low molecular weight. The differences found were small and inconstant and could be considered negligible.

The month of lactation did not seem to influence the proportion of soluble fatty acids (i. e., fatty acids of low molecular weight).

HIGGINS, Boston.

SUBMAMMARY TEMPERATURE AS A CRITERION OF LACTATION. M. ZELIC and Z. DJOKIE, München. med. Wchnschr. **80**:1238 (Aug. 11) 1933.

Taking the submammary temperature during lactation is the simplest method for determining the functional capacity of the breasts. The nearer the submammary temperature approaches the rectal or oral temperature, the better is the prognosis for lactation. If the determinations of temperature indicate a good prognosis, the mother should be advised to continue nursing even though she states that she has very little milk. Nursing does not seem to affect the submammary temperature.

BRAHDY, Mount Vernon, N. Y.

INFECTIONS IN RATS AND DEFICIENCY OF VITAMIN A. P. ARONS and M. P. J. VAN DER RYST, Nederl. tijdschr. v. geneesk. **76**:5445 (Nov. 26) 1932.

As a result of 276 autopsies and microscopic examination of 256 organs of rats, it appeared that infection is always found in the same organs, while it never occurs in others, and that infection generally occurs in a certain definite sequence in the various organs. When the organs were thoroughly examined, the infection

always proved to be accompanied by keratinization. Keratinization without infection was often found, but infection without keratinization did not occur. The accumulation of the products of secretion resulting from keratinization of the glandular ducts, together with the masses of keratin, produces a favorable substrate for bacteria. Arons and van der Ryst come to the conclusion that infections resulting from a deficiency of vitamin A are probably the result not of diminished resistance but of local changes caused by keratinized epithelium and atrophy of the glands.

VAN CREVELD, Amsterdam, The Netherlands.

Prenatal Conditions

COARCTATION OF THE AORTA AND CONGENITAL HYPERTROPHY OF THE HEART. J. HAROLD ROOT, Arch. Pediat. **50**:414 (June) 1933.

Attention is called to the rarity of this condition and the uncertainty of its etiology. The trend of opinion among more recent writers inclines toward a developmental origin, with heredity as a factor of importance in some cases.

A case of coarctation of the aorta with hypertrophy is reported in which the child died at the age of 13 days. The case report includes a complete record of the autopsy observations. A second case of congenital enlargement of the heart is described in which autopsy was not permitted.

WADDELL, JR., University, Va.

HYPERTELORISM. A. COWAN and M. SILBERMAN, J. Pediat. **3**:498 (Sept.) 1933.

Hypertelorism in a girl, 12 years old, and in her brother, 6 years old, is reported. Their mother and maternal grandfather showed the same abnormality. There was normal intelligence. The chief characteristic of the condition is the wide separation of the eyes, which is due to the large size of the lesser wings of the sphenoid bone. Strabismus is often present, and occasionally mental deficiency.

LEMASTER, Evanston, Ill.

HYPERTELORISM WITH CLEFT PALATE. W. R. BETT, Proc. Roy. Soc. Med. **26**:1016 (June) 1933.

A case of hypertelorism with cleft palate in an infant 7 days old is reported.

WILLIAMSON, New Orleans.

ACHONDROPLASIA WITH KYPHOSIS. ERIC PRITCHARD, Proc. Roy. Soc. Med. **26**:1019 (June) 1933.

A child, 12 months old, presented the typical appearance of well marked achondroplasia. During the fifth month an unusual feature—a kyphosis—developed in the dorsolumbar region. A roentgenogram of the spine suggested that the cause of the kyphosis might have been in the first lumbar vertebra and possibly also in the second. Perhaps it would be an exaggeration to say that they were "wedge-shaped," but they were not normal.

WILLIAMSON, New Orleans.

OCULAR SYMPTOMS IN HEREDITARY AND FAMILIAL CRANIOFACIAL DYSOSTOSIS. J. BADOT, Ann. d'ocul. **169**:833 (Oct.) 1932.

The ocular symptoms observed in hereditary and familial craniofacial dysostosis are bilateral exophthalmos and divergent strabismus, permanent or intermittent. Optic neuritis may develop and result in optic atrophy. In selected cases a decompressive trephine operation may be of value. Myopia, hyperopia, astigmatism and disorders of color vision are sometimes present. The recognition of the milder forms of dysostosis is more important for purposes of differential diagnosis than for therapeusis.

BERENS, New York. [ARCH. NEUROL. & PSYCHIAT.]

A Rare Anomaly of the Duodenum (Adhesions of the Second and Fourth Portions with the Duodenojejunal Angle to the Right of the Lumbar Column). J. Surmont, Bull. et mém. Soc. de radiol. méd. de France **19**:159 (April) 1931.

This rare anomaly occurred in a woman 30 years of age, who had complained for a long period of vague dyspeptic disorders. The interesting feature is that the duodenojejunal angle, which is considered to be one of the few fixed points of the abdominal cavity, was displaced to the right. There was no other congenital anomaly associated with this unusual condition.

Stulik, Chicago.

Chemical Constitution of Fetal Blood. G. de Toni, Arch. ital. di pediat. e puericult. **1**:197 (Dec.) 1932.

This is a monumental review of the subject of fetal blood chemistry and includes a bibliography of four hundred titles. The only hitherto unpublished material is the author's finding in the analysis of the cord blood of ten infants. He presents his own results, together with those of other students of this subject, in an easily available series of tables, which contain values for the following: acid-base balance, gases in the blood, dried residue, ash, total and fractional protein, nonprotein nitrogen, urea, aminonitrogen, uric acid, preformed and total creatinine, total fats, cholesterol, lecithin, dextrose, lactic acid, chlorine, calcium, magnesium, sodium, potassium, iron, phosphorus and sulphur.

Dooley, Kent, Conn.

Obliteration of the Urethra in the New-Born. R. Bertoni, Prat. pediat. **11**:138 (April) 1932.

The author describes an infant born with complete obliteration of the urethra. Satisfactory function of a surgically constructed urethral passage was obtained within two months after birth.

Dooley, Kent, Conn.

A Case of Partial Aplasia of the Right Ribs and Syndactyly of the Corresponding Hand. Gino Macchioro, Radiol. med. **18**:37 (Jan.) 1931.

A child 2 years of age was brought to the hospital with ordinary bronchitis. The child was mentally normal for its age. The right hand was much less developed than the left, and there was a syndactyly between the second and third and the third and fourth fingers. There was a marked depression, roughly triangular in form, in the right side of the wall of the chest. A roentgenogram showed the second, third, fourth and fifth ribs lacking from the posterior axillary line to the midclavicular line. There was slight scoliosis of the spinal column. No changes were evident in the skull or teeth.

The author thinks that this anomaly was due to pressure of the child's fist on the wall of the thorax during the early months of intra-uterine life. This would explain the anomalies of both the hand and the ribs.

Stulik, Chicago.

Craniofacial Dysostosis. R. Hirschfeld and P. Hirsch-Mamroth, Klin. Wchnschr. **10**:167 (Jan. 24) 1931.

The clinical picture of craniofacial dysostosis, as described by Crouzon in 1912, consists in cranial deformity, facial deformity and ocular changes in which there are exophthalmos, strabismus and ametropia. The condition appears to have a hereditary basis. The authors report a case and describe the roentgen findings in the skull. They point out that the photographs of patients with this condition give the impression that all patients are members of a single family. Their patient had a twin sister who was said to be normal, but they established that her skull was smaller than normal. The ocular changes are due to deformities of the orbits,

which give rise to marked exophthalmos. Many of the patients complain of headache for years, and in many instances there is abnormality of the sense of smell. The diagnosis has to be differentiated primarily from oxycephaly (Turmschädel). In the latter condition the abnormal height of the vault of the cranium is brought about by premature ossification of the coronary suture and, secondarily, of the sagittal and frontosphenoid sutures. This limits the growth of the frontal and temporal bones and the base of the skull, so that a compensatory height is brought about. Obviously, the two conditions have many points of similarity. Whenever pronounced changes are present in the skeleton of the face, craniofacial dysostosis is probably more likely. The conditions are probably closely related in regard to the features of their pathologic development. SILVERMAN, Syracuse, N. Y.

REPORT OF A CASE OF CONGENITAL ATRESIA OF THE GALLBLADDER OF A SIX MONTH OLD BABY. PIOTR SZUMOWSKI, Pediatria polska **12**:457, 1932.

The author states that there have been two cases of this type in his clinic within ten years. This shows the rarity of the condition. The boy whose case is reported was born at full term by normal delivery and was breast fed; he was the mother's fifth child.

At about the age of 3 weeks the skin became yellow and the stools were of clay color. The infant became progressively worse and died at the age of 6 months.

Feer reported a case in which the infant lived for ten months. The treatment is paliative. The diagnosis in these cases was proved by autopsy.

KRUPINSKI, Chicago.

CLEIDOCRANIAL DYSOSTOSIS. T. FREDBÄRJ and E. FAGERSTRÖM, Acta pædiat. **14**:321 (April 28) 1933.

The cases of four sisters are described, each of whom showed late or deficient closure of the fontanels, evidence of increased intracranial pressure, diminution or loss of visual acuity associated with optic atrophy, distended veins of the scalp, absence or deficiency of the hair and eyebrows, obstruction of the nasal passages, and failure of eruption of the teeth. Some showed, in addition, cervical ribs, genu valgum, dwarfism, mental retardation and other defects. The clavicles were normal. It is difficult, if not impossible, to classify these cases in any familiar category either of congenital malformations or of endocrine dyscrasias. The authors regard them as being related to cleidocranial dysostosis, of which cranial forms with normal clavicles have been described; but they incline to the view that there is a coexisting endocrine imbalance. MCINTOSH, New York.

CONGENITAL CYSTS OF THE LUNGS. A. DETHMERS, Acta radiol. **12**:135 (April 15) 1931.

A woman, aged 35, was sent to the hospital with a condition diagnosed as tuberculosis of the lungs. Sagittal and frontal roentgenograms showed the whole upper lobe of the right lung filled with small circular shadows, distributed irregularly; they were sharply circumscribed and enclosed air. One of the cavities showed a fluid level. The ring shadows were seen only in the upper lobe of the right lung. The other lobes and the heart, mediastinal organs and diaphragm were normal. It seemed probable from this picture that the upper lobe of the right lung was filled with congenital cysts, which diagnosis was confirmed by filling the cysts with iodized poppy-seed oil 40 per cent. The patient was kept under observation for a while. Her temperature remained normal, the expectoration decreased, and she was discharged in good condition. STULIK, Chicago.

Acute Infections

PRIMARY PERITONITIS COMPLICATING SCARLET FEVER. FERDINAND G. KOJIS and EDWARD J. MCCABE, Am. J. M. Sc. **185**:710 (May) 1933.

Three cases of primary peritonitis developed in 5,500 patients with scarlet fever at the Willard Parker Hospital, New York. Reports are given. A tabulated review of the literature shows that the condition is rare and of grave prognosis.

HENSKE, Omaha.

PRELIMINARY REPORT ON THE USE OF WILLOW BARK IN THE TREATMENT OF MALARIA. W. H. ZEIGLER, J. South Carolina M. A. **29**:30 (Feb.) 1933.

It was reported that a number of people had been cured of swamp fever by drinking tea made from willow bark, and it was suggested to the author that this bark be analyzed and compared with quinine. The main constituent of willow bark is salicin. A solution of willow bark slowly developed the movement of Paramecia and eventually killed them. The treatment is now being tried clinically in hospitals to determine the effect on patients with malaria, and a further report will be made giving the results.

ARSTON, Columbia, S. C.

ELECTROCARDIOGRAPHIC OBSERVATION IN DIPHTHERIA. JAMES M. FAULKNER, M. Clin. North America **16**:993 (Jan.) 1933.

Myocarditis in the course of diphtheria exhibits two phases in its development: first, a toxic parenchymatous degeneration of the conducting and contractile tissues, and second, a reparative inflammation called forth by the injured tissue. If the primary injury has been extensive, the degree of reparative inflammation is correspondingly increased and may be so intense as to choke off the nutrition of the healthy fibers which remain. The first phase does not usually produce recognizable clinical or electrocardiographic signs. The second phase, when associated with clinical signs, invariably brings about changes in the electrocardiogram indicating widespread damage involving the auricle, ventricle and conducting tissues. The value of such information in determining the prognosis and in estimating the extent of cardiac damage is readily appreciated. Four cases are described to illustrate the type of information which this method of examination may furnish. The electrocardiographic findings in these cases may be summarized as follows.

CASE 1.—A boy, aged 17, was admitted to the hospital on the fifth day of the illness. On the sixth day the heart rate dropped to 48. Electrocardiograms showed simple sinus bradycardia with normal conduction throughout. There was probable depression of the sino-auricular pacemaker due to vagal inhibition. There was no involvement of the myocardium. The patient recovered.

CASE 2.—A 3 year old boy showed symptoms of cardiac irregularity on the ninth day of the illness. The electrocardiogram at this time indicated depression of the sino-auricular pacemaker without interference with conduction. Later there was evidence of involvement of the ventricular myocardium. After the fifteenth day the patient's condition improved; there was a return of the intraventricular conduction time to normal, with an increase in the amplitude of the ventricular complexes. Death occurred later as a result of extensive paralysis, including the diaphragm.

CASE 3.—A 4 year old girl presented evidences of cardiac involvement on the ninth day of the illness. The electrocardiogram showed, successively, complete auriculoventricular dissociation, low voltage left bundle branch block, auricular flutter and auricular standstill, with only sporadic auricular contraction and auriculoventricular nodal rhythm. There was a return of sinus rhythm on the fifteenth day, but the patient died on the same day, apparently of pneumonia.

CASE 4.—Daily electrocardiograms were taken on a 26 year old man. The first change noticed corresponded in time with the development of clinical signs of cardiac involvement on the tenth day of the illness. At this time prolongation of the P R interval was noted. Later there appeared alterations in the ventricular complexes, consisting of a high take-off of the T wave in leads II and III, with a low take-off in lead I, followed by inversion of the T waves in all leads with upward convexity of the S T interval and diminution in amplitude of the Q R S complexes. Clinical recovery followed, with slower improvement in the electrocardiographic signs. The last record was taken about one year after the onset of the diphtheria. This showed a high take-off of T_2 and a prolonged P R interval. The patient was at work and had no symptoms of cardiac disturbance.

EUGENE SMITH, Ogden, Utah.

TRENDS IN DIPHTHERIA MORTALITY. EDWARD A. LANE, Pub. Health Rep. **48**: 473 (May 5) 1933.

In nine of ten states in which it was studied, the death rate due to diphtheria was from 36 to 47 per cent less during the period from 1900 to 1927 than it was during the years from 1895 to 1911. The yearly decrease in the mortality from diphtheria has been less in recent years than formerly.

LEMASTER, Evanston, Ill.

DIPHTHERIA IMMUNIZATION IN INFANCY. J. GRAHAM FORBES, Practitioner **130**: 587 (May) 1933.

In comparing the immunization records in England and the United States, Forbes notes that during the four year period ending Jan. 1, 1933, more than 680,000 children were immunized in New York City. Of these immunizations, 40 per cent were done in the health department stations, 32 per cent in schools and 28 per cent by private physicians. The total represents more than 40 per cent of the population under the age of 15. Of the children immunized, 230,000 were under 6 years of age and represent 36 per cent of the total preschool population, i. e., 635,000. In contrast, the available figures in England show that at the end of 1932 the number of children immunized or known to give negative reactions to the Schick test was 18,000, or 1.9 per cent of the children in London under the age of 15. Of this group, 7,000, or 40 per cent, were children of preschool age, but these represented only 3 per cent of the preschool population.

The author points out the importance of concentrating on the preschool group. He quotes from Godfrey's study of cities in New York State with a population of from 30,000 to 500,000, where it was shown that although from 50 to 70 per cent of children of school age had been protected, it was not until more than 30 per cent of those under 5 years of age had been immunized that any fall in the death rate from diphtheria for the community was produced. In New York City, in 1928, there were 10,776 cases, with 642 deaths, and in 1932 there were 3,585 cases, with only 212 deaths.

The author agrees with W. H. Park that the time for carrying out immunization against diphtheria should be during the second six months of the first year of life.

SAPPINGTON, San Francisco.

DIPHTHERITIC HEMIPLEGIA. L. BABONNEIX and A. MIGET, Gaz. d. hôp. **106**: 501 (April 5) 1933.

Hemiplegia occurs once in 1,500 cases of diphtheria; the onset is usually sudden, and complete recovery is rare. Left hemiplegia suddenly developed in a child, aged 29 months, on the eighteenth day after infection with diphtheria, in spite of the administration of 40 cc. of serum daily for the first eight days. Two

days later the right side became involved, and the patient died. Permission for autopsy was not obtained. Armand-Delille published the report of a case with a similar clinical course in which autopsy showed arteritis in the brain.

KAJDI, Baltimore.

THE SUPPOSED INFLUENCE OF CHICKENPOX ON TUBERCULOSIS IN INFANTS. BERNARD, LAMY and DUMANS, Presse méd. **40**:1429 (Sept. 21) 1932.

The effect of chickenpox on the development and course of tuberculosis has caused much speculation. Many writers have blamed it, not only for aggravating pulmonary tuberculosis, but for precipitating tuberculous meningitis or generalized tuberculosis. Hundreds of cases have been reported in which a latent or quiescent tuberculosis was activated by chickenpox with fatal results. The authors believe that this idea is greatly exaggerated, and that other factors, such as nutritional disorders, vomiting and superimposed infections, have more bearing on the course of the disease than does chickenpox. They report four cases of chickenpox in tuberculous children. Two of the patients died; the other two are in good condition. In no way could any connection be established between the course of the disease and the chickenpox, and the deaths could not be attributed to any activating influence of the virus. The conclusion is that the facts do not warrant the theory that infection with chickenpox is disastrous to the tuberculous child.

BIEKER, St. Petersburg, Fla.

DIFFUSE SEPTIC CELLULITIS OF ERYSIPELOID FORM DUE TO STAPHYLOCOCCUS. H. L. ROCHER and R. GUÉRIN, Rev. franç. de pédiat. **8**:548, 1932.

Three fatal cases are described. The initial lesion occurs on the face as an acne nodule. This nodule becomes inflamed and edematous and spreads. The final stage is an elevation in temperature and septicemia. Staphylococci can be isolated from the fluid. Surgical drainage is of no benefit.

SANFORD, Chicago.

SCARLET FEVER AND RHEUMATISM. V. MOLTSCHANOW, Rev. franç. de pédiat. **8**:675, 1932.

The author discusses the complication of rheumatism following scarlet fever and presents the clinical histories of fifteen cases. In his clinic at Moscow, rheumatism developed in from 8 to 10 per cent of the patients with scarlet fever. Moltschanow thinks that scarlatinal synovitis may be a manifestation of toxemia, for many cases of polyarthritis following scarlet fever show no cardiac lesions. Many of the cases do not develop for from six to ten months after scarlet fever. These findings justify a revision of the ideas of the "scarlet fever heart."

SANFORD, Chicago.

THE EXACT TIME OF THE INCUBATION PERIOD OF CONTAGIOUS DISEASES. H. BROKMAN, Pediatria polska **12**:127, 1932.

It was formerly thought that the incubation period represented the period from the time that the germ is deposited in the body to the time when it commences to produce symptoms of the disease, that is, when a sufficient amount of toxins develops to produce symptoms of the disease; but since the research work of Pirquet, Schick and Moro this theory has been changed. Pirquet in his research found that toxins may be produced in two ways: (1) by the development of the germ itself and (2) by the development of allergy due to the presence of the germ in the body. Therefore, he classifies diseases into two groups: 1. In the group in which allergy develops are included smallpox, measles, chickenpox and erysipelas. In this group the incubation period is from eleven to fifteen days. He called these diseases "Reaktionskrankheiten." 2. In the group in which the

toxins are produced by the germ itself he puts diphtheria, tetanus and scarlet fever. As the reaction in these diseases is due to the production of toxins in the body, the incubation period is not definitely known; it is, however, usually short, from two to four days.

KRUPINSKI, Chicago.

PATHOGENESIS OF POSTVACCINAL ENCEPHALITIS. H. BROKMAN, M. BUSSEL and M. HAYZUER, Pediatria polska **12**:130, 1932.

The pathogenesis and etiology of postvaccinal encephalitis are still unknown. Some authors claim that the condition is due to the smallpox virus itself. Others claim that it must be due to some other factors. The English commission for the study of such questions claims that the smallpox virus is the cause of postvaccinal encephalitis because the vaccination reduces the resistance of the nervous system to such an extent that even a small amount of virus might produce inflammation of the brain. This the authors have proved more or less experimentally, but no definite conclusion has been drawn as to either the etiology or the pathogenesis of the condition. Further research is in progress.

KRUPINSKI, Chicago.

TERTIAN MALARIA AS A CAUSE OF PURPURA HAEMORRHAGICA. P. C. KORTEWEG, Nederl. tijdschr. v. geneesk. **77**:1011 (March 4) 1933.

The author describes a case of hemorrhagic purpura observed during the acute stage of benign tertian malaria accompanied by thrombopenia, which is known as the essential symptom of this disease. The commencement and disappearance of the purpura were accurately established. This complication of malaria is rare, and study of the literature revealed only isolated cases.

VAN CREVELD, Amsterdam, The Netherlands.

Chronic Infections

THE CLINICAL SIGNIFICANCE OF CHOROIDAL TUBERCLES. R. T. PATON, Ann. Int. Med. **5**:997 (Feb.) 1932.

Choroidal tubercles occur as (1) granulomas or solitary tubercles and (2) miliary tubercles. The former type is in most cases associated with chronic tuberculosis, the latter with acute miliary tuberculosis. The usual number of tubercles found in one eye is three or four, but some observers have reported as many as seventy. They have been reported in from 14.6 to 100 per cent of the cases examined. Failure to find tubercles in tuberculous meningitis is probably due to difficulty in examination (tubercles in these cases are usually found only in the terminal stages) and in failure to make repeated and careful examinations. Both eyes should be examined. The presence of choroidal tubercles is often a valuable diagnostic aid. The prognosis is nearly always unfavorable; usually there is meningitis. The diagnosis is made from the various malignant growths. A case of tuberculous meningitis with choroidal tubercles is reported.

WILSON, Norfolk, Va.

PREVENTIVE VACCINATION WITH B C G ADMINISTERED BY MOUTH TO NONALLERGIC ADOLESCENTS AND ADULTS. M. A. MARFAN, Gaz. d. hôp. **106**:371 (March 11) 1933.

In equatorial Africa, 149 boys and girls were vaccinated by mouth with from 5 to 10 cg. of B C G. Three months later, 55 per cent of the subjects reacted positively to tuberculin administered intracutaneously; the remainder were revaccinated with the same dose of B C G, and a further 10 per cent then gave positive reactions.

In the hospital school for tuberculous children at Oslo, during the period from 1927 to the present, 253 nurses in training who gave negative reactions to the

Pirquet test were vaccinated with a single subcutaneous dose of B C G. Of the 104 who finally gave positive reactions to the tuberculin test, only 3 acquired the disease, whereas in the previous period, during which they were not vaccinated, 29.6 per cent of the nonallergic nurses acquired tuberculosis. The morbidity was 2 per cent for 176 medical students who were allergic, 2.3 per cent for 90 students who were vaccinated with B C G and 5.6 per cent for 82 nonallergic, nonvaccinated students.

KAJDI, Baltimore.

PROPHYLACTIC MEASURES AGAINST THE HEMATOGENOUS DISTRIBUTION OF PRIMARY TUBERCULOUS INFECTION IN CHILDHOOD. H. FERNBACH, Gesundhfürs. f. d. Kindesalt. **8**:37 (Feb.) 1933.

It is generally recognized that the prognosis of a primary tuberculous infection in childhood becomes increasingly serious in inverse proportion to the age of the child. Therefore, the best measures of prophylaxis are those which prevent infection in infancy. This can be done either by removing the child from the source of infection or by vaccinating with Calmette's bacilli, which represent a strain of low virulence, or with the vaccine of Bessau and Langer, which represents dead virulent tubercle bacilli.

In general neither of these measures has won great popularity; hence one must still cope with infection in infancy and in early childhood. The primary complex is probably not the most dangerous feature of tuberculous infection at this age; it is rather the hematogenous distribution, which may occur at any stage in the infection and which increases in frequency in inverse proportion to the age of the child. The physician has no specific measures by which he can strengthen the defense against this scattering of the infection throughout the body. It is probable that the nonspecific measures which have long been in use, such as change of climate and open air therapy, are valuable measures of prophylaxis. For economic reasons, however, these measures can be used in only a small percentage of cases.

Bessau has called attention to the parallelism between seasonal incidence of rickets and spasmophilia and that of miliary tuberculosis and tuberculous meningitis. He has suggested that in both cases the lack of sunlight causes some defect in the mesenchymal tissues which in the case of tuberculosis permits a generalization of the disease. He also calls attention to the fact that there is a negative calcium balance in active tuberculosis. On purely empirical grounds, ultraviolet rays and cod liver oil have been used in the therapy of tuberculosis. Fernbach believes that these measures should not be restricted to children suffering from manifest tuberculous disease, but should be carried out in the case of every child who has a tuberculous infection. He points out that generalization usually occurs early in the course of an infection. In eight years about 900 children suffering from tuberculosis were treated in the children's clinic at Leipzig. Of these children, 325 entered the hospital suffering from tuberculous meningitis or miliary tuberculosis; of the remaining 663 children, only 8 contracted miliary tuberculosis. Hence, if prophylaxis is to be of value, it must be used as soon as a tuberculous infection is recognized.

It has been the practice of Fernbach to give to all the children who enter the dispensary with signs of a primary infection cod liver oil and viosterol and, in the winter, treatment with ultraviolet rays. He suggests that if this regimen were carried out in a large group through several years, the value of this prophylaxis might be determined.

REICHLE, Cleveland.

Internal Diseases

PRIMARY TUMORS OF THE OS CALCIS. B. L. COLEY and G. S. SHARP, Am. J. Cancer **16**:1053, 1932.

The os calcis is an infrequent site of a primary bone tumor; but thirty-two cases were found in a combined study of the records of the Bone Tumor Department of Memorial Hospital and of the Bone Sarcoma Registry of the American

College of Surgeons. Indefiniteness of clinical manifestations and of roentgenographic findings makes a positive preoperative diagnosis extremely difficult. An accurate diagnosis is essential for correct treatment. It follows that this can be obtained in most cases only by histologic study. If aspiration biopsy is inconclusive, as it often is, a surgical biopsy is indicated. In osteogenic sarcoma the treatment is early amputation. We do not favor prolonged irradiation preliminary to amputation, but, if adequate radiation can be delivered in a brief period, followed promptly by amputation, we see little objection to its employment; we have no data to suggest that the results are thereby improved. Endothelial myeloma, being an extremely radiosensitive tumor and characterized by early and wide dissemination to other bones as well as the lungs, is best handled by irradiation with high voltage roentgen rays and injections of mixed toxins of erysipelas and Bacillus prodigiosus. A giant cell tumor may be treated successfully by irradiation alone. There are, however, a number of cases of tumors in other bones in which irradiation has been used in the past in doses that have caused permanent damage to the skin, soft parts and bone. The response to irradiation varies considerably in individual cases of giant cell tumors. Thorough curettage, followed by an escharotic, such as zinc chloride or phenol and alcohol, is an established method of dealing successfully with these cases, particularly when the tumors are well defined and have not broken through the cortex and extended into the soft parts. The combined case records of the Memorial Hospital and the Bone Registry show that an early, correct diagnosis was seldom achieved, and that there was no well conceived plan of treatment for primary tumors of the os calcis.

AUTHORS' SUMMARY. [ARCH. PATH.]

LIPOID HISTIOCYTOSIS: NIEMANN-PICK'S DISEASE WITH LEUKEMOID BLOOD REACTION. CHARLES F. FISHER, Arch. Pediat. **49**:574 (Sept.) 1932.

The bibliography of lipoid histiocytosis is reviewed, and a case with a leukemoid blood picture is presented. The condition was suspected because of splenohepatomegaly, vacuolated lymphocytes in the circulation and cherry-red spots in the maculae. The diagnosis was confirmed by splenic puncture.

WADDELL, University, Va.

GIARDIASIS IN YOUNG CHILDREN. JOHN ZAHORSKY, Arch. Pediat. **49**:627 (Sept.) 1932.

The writer records his further observations on giardiasis as a cause of obscure gastro-intestinal symptoms, reporting eight more cases. He believes that, particularly in infants, frequent stools, a lessening of food absorption and retardation in growth can at times be the result of infestation with giardiae. He feels that the administration of bismuth salicylate several times a day in full doses will correct the apparent gastro-intestinal disturbance.

WADDELL, University, Va.

PERINEPHRITIC ABSCESS IN CHILDREN. LEON ANTELL, Arch. Pediat. **49**:743 (Nov.) 1932.

Three cases of perinephritic abscess in children are reported, each one of which presents different problems and different manifestations. The author reviews the condition, particularly from a pediatric angle, and points out the more common errors in differential diagnosis. Perinephritic abscess of extrarenal origin is thought to be a metastatic infection caused by staphylococci in 80 per cent of the cases, peripheral infections, such as boils and paronychia, serving as portals of entry. Antell points out that while acute cases may be comparatively easy to diagnose, subacute and chronic cases may be confused with pulmonary, abdominal and spinal diseases and disease of the hip joint, the diagnosis at times being difficult.

The most important aids in diagnosis are a good history, particularly as regards peripheral infections, and positive roentgenologic findings, the possibility of this condition always being borne in mind. The results of examination of the urine and blood and the cystoscopic findings are of less importance.

WADDELL, JR., University, Va.

RESPIRATORY INFECTIONS IN CHILDREN. HENRY HEIMAN, Arch. Pediat. **50**:53 (Jan.) 1933.

This article presents nothing new.

HYPERTHYROIDISM IN CHILDREN. R. S. DINSMORE, J. A. M. A. **99**:636 (Aug. 20) 1932.

The author is led to believe that hyperthyroidism is more frequent in children than was formerly thought. A review of fifty-seven cases is given. The symptoms are similar to those found in thyroid disease in adults. Estimations of the basal metabolism have been generally unsatisfactory. The surgical problems are those encountered in the adult, except that greater care should be exercised to leave enough tissue for the normal requirements of a child. The tendency for keloid formation in the scar is nearly universal. The grenz rays give satisfactory end-results. The outcome of the operation seems to be satisfactory over the course of a few years. Occasionally, transient hypothyroidism develops.

BONAR, Salt Lake City, Utah.

INTESTINAL STASIS IN INFANTS AND CHILDREN. C. G. KERBY, J. A. M. A. **99**:1062 (Sept. 24) 1932.

The term intestinal stasis indicates dysfunction due to faulty intestinal mechanics. For normal emptying of the stomach there must be normal elimination by the bowels. The colon is in most cases the primary seat of the disturbance. Occasionally, intestinal bands are the cause. The following three groups are recognized: (1) persons with an elongated, redundant, sacculated colon and sigmoid; (2) those in whom stasis is dependent on muscle incoordination and is evidenced by circular fiber spasm, without demonstrable lesions, and (3) those having symptoms identical with the symptoms in group 2 in whom in the abdominal cavity, within or without the intestinal tract, there is a pathologic process such as mesenteric adenitis, mucous colitis or chronic appendicitis. Treatment consists in the removal of a diseased appendix and the administration of atropine and liquid petrolatum for those with nerve imbalance (group 2).

BONAR, Salt Lake City, Utah.

AN IMPROVED COSEASONAL THERAPY. WARREN T. VAUGHAN, J. Allergy **3**:542 (Sept.) 1932.

The author recalls some of the beneficial effects of coseasonal treatment of patients with hay fever by administration of small doses of the specific extract. In 1931, he treated fifteen patients somewhat similarly, with good results. At the start he gave 10 or 20 pollen units subcutaneously daily, increasing the dose by 20 pollen units daily, usually to from 80 to 100 units, until relief was obtained. The interval between doses was then gradually increased as long as the hay fever persisted. On the return of symptoms the interval was shortened, but the dosage was not increased.

The perennial method is still considered the method of choice, but if it does not permanently desensitize, it is questioned whether the inconvenience of treatment throughout the year justifies the slight increase in efficacy.

HOYER, Cincinnati.

THE SPECIFIC DYNAMIC RESPONSE TO PROTEIN OF INDIVIDUALS SUFFERING FROM DISEASE OF THE HYPOPHYSIS. MARGARET W. JOHNSTON, J. Clin. Investigation **11**:437 (March) 1932.

Persons with definite abnormality of the pituitary body showed a large increase in the production of heat with the ingestion of protein, but the evidence does not support the theory that the pituitary gland is responsible for the specific dynamic action of protein.

KUGELMASS, New York.

PURPURA HEMORRHAGICA: TREATMENT OF A CASE WITH INTRAMUSCULAR INJECTION OF WHOLE BLOOD. ELLIS POLIAKOFF and G. A. NEUFFER, J. South Carolina M. A. **29**:91 (April) 1933.

A white boy, aged 5 years, had thrombocytopenic purpura which evidently was caused by an infection in the upper nasal passages. Despite treatment with calcium, iron and ergot, the patient continued to have purpuric spots; however, when repeated intramuscular injections of whole blood were given, the patient showed immediate improvement, and there was no evidence of recurrence of the purpura.

WESTON, JR., Columbia, S. C.

PRIMARY SARCOMA OF THE LIVER IN A SEVEN MONTH INFANT. LEONARD F. BENDER, M. J. & Rec. **137**:405 (Nov. 16) 1932.

As tumors of the liver of any type must be considered a great rarity in infancy and childhood, the author felt that a case he observed should be reported. The report is accompanied by observations at autopsy and histopathologic studies.

WILLIAMSON, New Orleans.

NONDIPHTHERITIC LARYNGOTRACHEOBRONCHITIS. S. KIRKPATRICK and S. M. KIRKPATRICK, South. M. J. **26**:287 (March) 1933.

The predominating organisms found in laryngotracheobronchitis are staphylococci and streptococci. Low tracheotomy with mechanical removal of mucus is essential in severe cases. Fluids should be forced. Antispasmodics are also useful. Laryngotracheobronchitis is a serious disease, with a mortality around 50 per cent, and it is often diagnosed as diphtheria. The condition is much more prevalent than was formerly thought. An early diagnosis should be made and energetic treatment instituted.

SCHLUTZ, Chicago.

THE PERIODIC GROUP OF DISORDERS IN CHILDHOOD. W. G. WYLLIE and BERNARD SCHLESINGER, Brit. J. Child. Dis. **30**:1 (Jan.-March) 1933.

The type of case discussed is variously referred to as one of bilious attacks, cyclic vomiting, ketogenic or acetonemic vomiting, recurrent pyrexial attacks or migraine, according to the predominance of one or another of the symptoms composing the syndrome. To the authors these terms appear to represent not so many clinical entities, but merely clinical variations in the expression of some common underlying factor.

The symptomatology of these clinical states is outlined, and it is shown that they bear a great similarity to one another; such distinctiveness as they possess they owe to a preeminence of one or another of the four chief symptoms, i. e., headache, vomiting, fever and abdominal pain.

In the eighty cases on which this report is based, vomiting was especially mentioned in sixty-three cases; a temperature from 102 to 105 F. during attacks was noticed in sixteen children; headache was complained of in sixty cases. Abdominal pain was present in some degree in most cases.

Accessory symptoms that were noticed in individual cases included pains in the limbs, urticaria, asthma, faintness, dizziness and lapses of consciousness. In several cases a raised blood pressure was found.

The possible allergic basis for these periodic attacks is considered, and the heredity factor is discussed briefly. The authors feel that neither their investigations nor those of others warrant the statement that ketosis is a primary factor in the production of periodic vomiting.

In the study of these periodic disturbances opinions as to their causation have principally concerned two systems, the hepatic and the nervous. From these investigations it is concluded that the primary factor underlying the occurrence of the periodic group of disorders is of nervous origin.

LANGMANN, New York.

THREE CASES IN WHICH AN ERUPTION OF PETECHIAE FOLLOWED ATTACKS OF VOMITING. E. W. GOODALL, Brit. J. Child. Dis. **30**:39 (Jan.-March) 1933.

Crops of petechiae developed in one child recovering from faucial diphtheria and in two others during an attack of scarlet fever. The author attributes the development to straining accompanying severe attacks of vomiting.

LANGMANN, New York.

THE SIGNIFICANCE AND ORIGIN OF LACTIC ACID IN THE GASTRIC CONTENTS. IAN MURRAY and ANDREW B. ROBERTSON, Brit. M. J. **1**:607 (April 2) 1932.

1. An investigation of test meals from fifty subjects, pathologic and normal, showed the presence of lactic acid in 74 per cent. While occurring more frequently in cases of hyposecretion, it may be present even in hypersecretion. The presence of lactic acid had apparently no clinical significance.

2. The production of lactic acid was almost certainly due to the action of Bacillus acidi lactici, which was found to be present in the normal stomach.

AUTHORS' SUMMARY.

DIABETES IN CHILDHOOD: SYMPOSIUM. Glasgow M. J. **118**:315, 1932.

Etiology, Diagnosis and Prognosis, by G. B. Fleming.—The etiology is obscure. Infection and heredity play important rôles. The author saw no patients under 1 year of age. Erroneous diagnosis is made in cases of gastro-enteritis in infants and in cases of disease of the central nervous system when glycosuria is present. Illustrative cases are reported. Renal diabetes is mentioned. In ketosis the blood sugar curve may simulate a diabetic dextrose tolerance curve. Of thirty patients thirteen are dead, nine have a decreased tolerance, and eight have improved. The cause of death in most instances was coma. Most of the deaths occurred in patients over 10 years of age, when the diet of the child is less liable to be controlled by the parents and dietary errors are found.

Treatment, by Noah Morris.—At first a low intake of food is prescribed until the urine becomes sugar-free. The increase to the caloric requirement may then be gradual or immediate. Insulin is given to control the excretion of sugar. The author advocates an adequate diet, 50 per cent above the basal caloric requirement, as the well fed child is less liable to steal food. The diet is based on dietary units of 120 calories, each made up of 5 Gm. of carbohydrate (20 calories) and 7.5 Gm. each of protein and fat (100 calories). The total number of calories prescribed is divided by 120, so that the diet can be ordered in terms of these units. The parents are instructed to keep the urine sugar-free. The blood sugar is checked. Infections decrease the efficacy of insulin.

Hypoglycemic reactions are treated by sugar orally or by epinephrine, 5 minims (0.3 cc.), hypodermically. Muscular exercise tends to decrease the blood sugar.

Coma, by Finlay J. Ford.—There is usually a history of headache, drowsiness, vomiting, abdominal pain and constipation. Oliguria is common. Albumin and casts are found in the urine. The blood shows dehydration, hyperglycemia and a decrease in carbon dioxide. Insulin is given hypodermically, and 10 per cent dextrose is given by mouth and by rectum. The authors never had to resort to intravenous injections of dextrose. Sodium bicarbonate is given, from 10 to 15 grains (0.7 to 1 Gm.) every four hours, until the coma is over. Warmth is applied, free action of the bowel is induced, and if there is vomiting, gastric lavage is performed. Infections make the prognosis less favorable. Prevention by proper control of the diabetic child is ideal.

Significance of the Blood Sugar Curve in Ketosis, by Mary L. Gilchrist.—Patients not suffering from diabetes were given a ketogenic diet, and the dextrose tolerance curve became the same as that of a diabetic patient. Thus, before making a dextrose tolerance curve, the previous diet should be taken into consideration.

SNELLING, Toronto, Canada.

ACUTE VOMITING AND DIARRHOEA IN CHILDREN. ALAN MONCRIEFF, Practitioner **130**:307 (March) 1933.

In this article, the author emphasizes at once that vomiting and diarrhea represent symptoms and not one disease or two diseases. From a practical point of view, the two symptoms occurring together may be under the following conditions: (*a*) when the disturbance is primarily in the gastro-intestinal tract, as in gastro-enteritis due to bacterial infection, when the cause is essentially irritation by food and when a prolonged dietetic error is followed by the acute onset of diarrhea and vomiting, and (*b*) when the etiologic factor is outside the gastro-intestinal tract, as is occasionally the case in pneumonia or in empyema, otitis media, mastoiditis, pyelitis or tonsillitis. If diarrhea and vomiting occur singly, they may be symptomatic of widely different diseases. Vomiting may result from intracranial disease or from an acute abdominal disorder, such as inflammation or obstruction. Also diarrhea, especially if blood is present, may be a sign of intussusception or rectal polyp.

Moncrieff lays emphasis on the following factors in treatment: (*a*) the need of skilled nursing, (*b*) the value of a short period of starvation early in the disease, (*c*) precautions for isolation in all doubtful cases and (*d*) the need of considering the patient as a whole and the avoidance of too great interest in one special symptom. Of first importance in the treatment of infants is the disturbance of water balance. The requirements for water and the means of administration are discussed fully. The author feels that the use of drugs is disappointing, although bismuth and sometimes opium are valuable in certain cases. As to dietary aids, diluted breast milk is advised. Later it is stated that the fat must be kept low, the protein must be given gradually and the sugar must be nonfermentable. Skimmed lactic acid milk containing dextrimaltose is recommended. SAPPINGTON, San Francisco.

ATELECTATIC BRONCHIECTASIS AND DISPLACEMENT OF THE HEART. A. G. MAITLAND-JONES, Proc. Roy. Soc. Med. **25**:1734 (Oct.) 1932.

A boy of 13 years had suffered from persistent cough since an attack of pneumonia eight years previously. Roentgenograms taken at this time showed heavy hilar shadows, with an increase of pulmonary markings at the base of the right lung. The diaphragmatic movements were fair and equal. A well defined shadow behind the heart was noted; it had the appearance of a collapsed lung. Two months later iodized poppy-seed oil 40 per cent was injected, and the child was turned on the left side. The iodized oil failed to enter the base of the left lung, although the right lung filled normally. Clubbing of the fingers was present. The percussion note was dull over the base of the left lung, and there was a small area of bronchial breathing below the angle of the left scapula. The apex beat of the heart was in the sixth interspace. WILLIAMSON, New Orleans.

TWO CASES OF ACCESSORY LOBE OF THE AZYGOS VEIN. E. A. COCKRAYNE, Proc. Roy. Soc. Med. **26**:148 (Dec.) 1932.

1. A girl of 3 years was admitted to the hospital on account of six attacks of asthma alternating with attacks of papular urticaria.

2. A boy of nearly 7 years was admitted to the hospital for investigation as to the cause of asthma of four years' duration.

The roentgenogram in each case showed an accessory lobe of the azygos vein.

WILLIAMSON, New Orleans.

RENAL RICKETS. E. A. COCKRAYNE, Proc. Roy. Soc. Med. **26**:153 (Dec.) 1932.

The author reports a case of renal rickets in a child of 10 years. The results of roentgen examination, the blood cytology and the blood chemistry are given.

WILLIAMSON, New Orleans.

BRONCHIECTASIS. T. STANLEY RODGERS, Proc. Roy. Soc. Med. **26**:155 (Dec.) 1932.

A girl of 6½ years had suffered from cough since an attack of croup at the age of 5 months. She had pneumonia at the age of 18 months and again at 23 months, and whooping cough at 4 years. The left side of the chest moved less than the right. The note at the base of the lung was dull; the breath sounds were tubular. On the right side, near the angle of the scapula, was an impaired note. The fingers showed slight clubbing. Roentgen examination showed an area of unresolved pneumonia in the lower lobe of the right lung and on the left side a triangular shadow with its short base on the diaphragm and its apex at the level of the body of the sixth dorsal vertebra. A roentgenogram taken after the injection of iodized poppy-seed oil 40 per cent showed the triangular shadow to contain bronchi crowded together and somewhat dilated.

WILLIAMSON, New Orleans.

CONGENITAL EPULIS (ITS HISTOGENESIS). P. MOULONGUET and GENEVIEVE DELAMBERT, Ann. d'anat. path. **9**:887, 1932.

In studying thirteen cases of the rare congenital epulis, the authors came to the conclusion that it is a form of benign adamantinoma, a sort of embryonic misplacement of nests of ameloblasts. The spongy cells surrounding the nests of ameloblasts are interpreted as degenerated groups of these cells, similar to the central degenerated spongy cells of ordinary adamantinomas but having an opposite polarity or distribution.

MELNICK, Chicago. [ARCH. PATH.]

LOBSTEIN'S DISEASE. BARBONNEIX, DUCROQUET and WILM, Bull. Soc. de pédiat. de Paris **30**:437 (July 12) 1932.

A boy, 11 months old, showed fractures of almost all the long bones of the body. No cases of osteogenesis imperfecta could be found in the family history. He had marked hypercalcemia. Deformities of the extremities were first noted when the infant was 2 months old. He was particularly susceptible to infection of the upper respiratory tract, and was admitted to the hospital because of acute bronchitis.

BENJAMIN, Montreal, Canada.

TREATMENT OF INFESTATION WITH OXYURIS. A. CHIODO, Prat. pediat. **10**:451 (Oct.) 1932.

The author reports the cases of three children who had long-standing infestations with Oxyuris vermicularis, which resulted in grave intoxication and anemia, despite treatment with the usual remedies. The children were cured quickly by the use of chiniofon by mouth (0.25 Gm. three times a day) and by rectum (from 0.25 to 1 per cent solution).

DOOLEY, Kent, Conn.

A CASE OF MARBLE DISEASE. G. GUASSARDO, Riv. di clin. pediat. **30**:1531 (Dec.) 1932.

Marble disease, which is called by the author ostéosclérose éburnisante érythémique, is a clinical condition in which there are an ivory-like ossification of the bones and an erythroblastic blood picture. It is congenital and sometimes familial.

A case is reported of a bottle-fed, well nourished boy 3 months old. He was taken to the clinic because of enlargement of the spleen. Another child in the same family had previously died at 11 months of age of a condition diagnosed as leukemia. A roentgenogram showed marked density of the bones, equivalent to that of a much older child.

Analysis of the blood showed 40,600 nucleated cells and 3,600,000 erythrocytes, with 58 per cent hemoglobin. The differential count showed 34 per cent erythro-

blasts, 10 per cent myelocytes, 11 per cent polymorphonuclear neutrophils, 2 per cent polymorphonuclear basophils, 21 per cent monocytes, 4 per cent transitionals, 14 per cent lymphocytes and 5 per cent megakaryocytes. The blood calcium was 9.5 mg. per hundred cubic centimeters, and the blood phosphorus, 4.9 mg. The red cells showed anisocytosis, poikilocytosis and polychromatophilia. The Wassermann and tuberculin tests gave negative results. Examination of the blood and bones of the mother and father demonstrated no abnormality. The child was followed up for six months.

The author believes that the condition is of neuro-endocrine origin, with effects on the peripheral vessels (vagotonia). HIGGINS, Boston.

THYMIC HYPERTROPHY AND ITS PATHOLOGIC SIGNIFICANCE. M. PÉHU, Arch. f. Kinderh. **95**:251 (Feb. 5) 1932.

The author gives a historical survey of the literature on thymic hypertrophy. At the present time much of it must be discounted, as it is now known that in any disease the thymus atrophies rapidly, and what was formerly considered to be an enlarged thymus was in reality the thymus in health, as revealed at autopsy. Angioma of the base of the tongue, macroglossia in myxedema or mongolism, and diseases of the larynx, especially congenital stridor and malformation of the epiglottis, have all led to the erroneous diagnosis of enlargement of the thymus. The author concludes that the pathologic rôle of so-called thymic hypertrophy is continually shrinking, and that its existence as a specific disease is now in question.

LESLIE, Evanston, Ill.

THE TREATMENT OF ENURESIS. K. STOLTE, Arch. f. Kinderh. **95**:294 (Feb.) 1932.

The author does not regard enuresis in itself as a manifestation of psychologic maladjustment, but he does believe that unsympathetic treatment of the condition, particularly blame or scorn, may lead to serious personality difficulties. He emphasizes the importance of encouraging the child and of building up his self-esteem and self-reliance. He has obtained good results by gradually lengthening the intervals during the day at which the bladder is emptied; he believes that this stretches the bladder and strengthens the sphincters. LESLIE, Chicago.

THE IMPORTANCE OF UROLOGIC EXAMINATION IN THE DIAGNOSIS OF PYURIA IN CHILDHOOD. ANTON VON ADLER-RAEZ, Arch. f. Kinderh. **98**:234 (Feb. 21) 1933.

Emphasis is placed on the importance of a systematic urologic investigation in all cases of pyuria in which the etiology is not clear or in which medical and dietetic management does not produce good results in a reasonable period of time. Two cases of pyuria are presented, illustrating the necessity of urologic procedures in establishing the correct diagnosis of the pathologic condition in the urinary tract so that proper treatment can be instituted. LEMASTER, Evanston, Ill.

UROLOGIC EXAMINATIONS IN CHILDHOOD. A. VIETHEN, Arch. f. Kinderh. **99**:9 (March 21) 1933.

The author presents his clinical impressions and views based on urologic examination of 150 children. Intravenous pyelograms are usually made first, and when necessary are supplemented by cystoscopy. Retrograde pyelography is much more disturbing to the child and is not without danger. In early cases of tumor of the kidney and renal tuberculosis it was found that hematogenous urography alone did not furnish sufficient information. LEMASTER, Evanston, Ill.

ECHINOCOCCUS CYSTS OF THE LIVER; THIRTY-FOUR CASES FROM THE CLINIC AT GÖTTINGEN FROM 1912 TO 1931. E. R. HYDEMANN, Deutsche Ztschr. f. Chir. **238**:345 (Dec. 30) 1932.

Between 1877 and 1890, nineteen cases of echinococcus cyst of the liver were reported from this clinic; between 1898 and 1907, thirteen, and between 1912 and 1931, thirty-four. The increase in cases is attributed to an increase in the number of patients seen rather than to an increase in the incidence of the disease. In this period of twenty years the thirty-four cases of echinococcus cyst of the liver reported and six echinococcus cysts of the lungs occurred in 70,000 patients, a total percentage of 0.057. The ratio of the percentage of primary hepatic cysts to that of primary pulmonary cysts, 85 to 15, corresponds to that of most statistics. The disease may be considered as of endemic origin, since only two patients were not born in South Hanover. Fifty-five and seven-tenths per cent of the series were females. The series included four children between 7 and 12 years of age and two youths of 15 and 16 years.

The author states that the disease usually remains unnoticed unless complications occur. These may be infection or rupture of the cyst or symptoms produced by enlargement of the cysts with pressure on the liver and adjacent organs, especially the stomach. Occasionally a patient consults a physician because of a painless swelling.

In uncomplicated cases of echinococcus cyst the diagnosis is usually easily made if the possibility of the disease is kept in mind. The presence of cysts in the liver is often proved by roentgenograms, and pulmonary cysts are sometimes disclosed in routine studies. Occasionally errors in diagnosis are made by reason of the roentgenogram, as in one case in which a diagnosis of calcification of the gallbladder was made. At operation a normal gallbladder was found, and near it a calcified echinococcus cyst which produced the symptoms. In thirteen cases in which the blood picture was studied, eosinophilia occurred seven times. In two cases there was an eosinophil count of 47 per cent and 50 per cent, respectively.

In 23.5 per cent of the cases the cysts were not infected and showed no signs of rupture. In these cases six patients were operated on twice. One patient had five cysts the size of a hen's egg. This case was considered inoperable. Another patient had a cyst the size of a child's head occupying the whole left lobe of the liver. The cyst was removed in its entirety, and the patient was discharged well in eleven days. Infected and calcified echinococcus cysts offer more difficulty from the surgical point of view because of the accompanying inflammation and the presence of adhesions. Rupture of a cyst into the biliary passages is a dangerous complication, especially if daughter cysts are present. A case is mentioned in which hundreds of cysts varying in size from that of a pea to that of a hen's egg were found on exploratory laparotomy. The case was considered inoperable. Seven years later, at the age of 56, the patient stated that he was in normal health and had gained 35 pounds (15.9 Kg.). One patient died of echinococcus cyst of the brain three years after a solitary cyst had been removed from the liver. Rupture of a cyst into the abdominal cavity may cause death by anaphylactic shock. Rupture of a cyst into the pleural cavity or subdiaphragmatic space was not observed. Injections of solution of formaldehyde may be tried in the case of inoperable simple cysts but are contraindicated in that of infected or calcified cysts.

The author believes that in general the prognosis of echinococcus cyst of the liver is good, especially in uncomplicated cases, in which the mortality was zero. In cases in which the cyst was infected or calcified, the mortality was 22.7 per cent. The cysts are multiple in at least a third of the cases, and thorough exploration as well as roentgenographic study is necessary to disclose all the cysts.

GLASER, Rochester, N. Y.

TREATMENT OF ANEMIA WITH AN ORGANIC SULPHUR COMPOUND. FRANK, Monatschr. f. Kinderh. **57**:23 (Feb.) 1933.

The author reports the results of treatment with an organic sulphur-containing compound in cases of anemia. When combined with ferrum reductum this compound gives good results.

GERSTLEY, Chicago.

THE LIPOID CHEMISTRY OF XANTHOGRANULOMATOSIS H. KLEINMANN, Virchows Arch. f. path. Anat. **282**:613, 1931.

Kleinmann made quantitative determinations of the lipoids of tissues from the case of xanthogranulomatosis reported by Ighenti (*Virchows Arch. f. path. Anat.* **282**:585, 1931; abstr., ARCH. PATH. **15**:433, 1933). His results agree in general with those of Chiari. The essential findings were: an increase in total lipoids due to a relative and absolute increase in cholesterol and its esters, a reversal of the ratio of cholesterol ester to cholesterol as compared with normal tissues, and a reversal of the ratio of cholesterol to lecithin as compared with Niemann-Pick disease. The ratio of cholesterol to lecithin was 5.85:1, as compared with 1:9.3 for the spleen in Niemann-Pick disease. The ratio of cholesterol ester to cholesterol was 5.08:1, as compared with from 1:2 to 1:4 for normal tissues. The total lipoids of the spleen were not increased, but the ratio of cholesterol to lecithin was altered, being 2.7:1. For the normal spleen this ratio is 1:1.8, and for the spleen in Niemann-Pick disease, 1:9.3. The ratio of cholesterol ester to cholesterol was 3.5:1. The total cholesterol of the liver was increased, being 3.86 per cent as compared with 1.28 per cent for the normal liver. The ratio of cholesterol to lecithin was 3.7:1. SCHULTZ, Evanston, Ill. [ARCH. PATH.]

PHOSPHATIDE CONTENT OF THE BRAIN IN NIEMANN-PICK DISEASE. E. EPSTEIN, Virchows Arch. f. path. Anat. **284**:867, 1932.

The child with Niemann-Pick disease may exhibit symptomatic evidence of cerebral involvement, amounting in some cases to idiocy. Histologically, the brain reveals the same process of lipoid storage present in other organs. The lipoid material, which is chiefly the phosphatide lecithin, is present in ganglion cells, periadventitial cells and glia cells. A case reported by Pick was clinically one of typical familial infantile amaurotic idiocy with the characteristic retinal findings. The anatomic picture was that of typical Niemann-Pick disease. Pick suggested that the Tay-Sachs type of amaurotic idiocy may be a form of Niemann-Pick disease. Epstein examined chemically the brain of a child with Niemann-Pick disease and with the clinical symptoms of idiocy, but without the characteristic retinal changes of Tay-Sachs disease. The lecithin of the brain was increased 80 per cent above the lecithin content of the brain of a normal child of the same age; the lecithin content of the spleen was increased a hundredfold. The total phosphatide of the brain was increased 90 per cent; that of the spleen, nineteenfold. Epstein concludes that there are two types of Niemann-Pick disease, one with, the other without, the retinal changes of infantile amaurotic idiocy. There is also a "pure" type of amaurotic idiocy, with retinal changes but without the characteristic alterations of Niemann-Pick disease in the liver, spleen and bone marrow. SCHULTZ, Evanston, Ill. [ARCH. PATH.]

RELATIONSHIP BETWEEN ARTICULAR RHEUMATISM AND TUBERCULOSIS. W. LOEWENSTEIN, A. STRASSER and A. WEISSMANN, Wien. Arch. f. inn. Med. **22**:229 (April) 1932.

Repeated efforts have been made to establish a connection between acute articular rheumatism and tuberculosis. C. Reitter and E. Löwenstein have recently demonstrated a tuberculous bacillemia in acute articular rheumatism, and have advanced the theory that this is a not infrequent form of manifestation of the tubercle bacillus.

Statistical data as to the frequency of combined tuberculous manifestations and acute articular rheumatism show that such a combination is extremely infrequent, the incidence being less than 1 per cent.

The authors obtained positive cultures of the blood in 34.4 per cent of their patients with disease of the joints. They do not believe that a causal relationship exists between tubercle bacillemia and disease processes characterized particularly by infectious arthritis.

Infectious arthritis develops on the basis of some systemic infection, which may be tuberculosis. Sensitization of the mesenchyme is responsible for the development of the symptoms.

SCHLUTZ, Chicago.

TESTS OF LIVER FUNCTION WITH GALACTOSE SOLUTIONS INJECTED INTRAVENOUSLY. STEPHAN POLLAK, Wien. Arch. f. inn. Med. **22**:317 (April) 1932.

The author has found that with the use of intravenous galactose solutions the blood sugar curve shows marked alteration in pathologic conditions of the liver. In the normal person it will reach values of 150 mg. on an average within fifteen minutes after the administration of this substance, an increase over the normal level between 42 and 70 per cent.

In cirrhosis of the liver the increase in values does not attain that in a normal condition and it fluctuates between 10 and 36 per cent. In cases of pernicious anemia the same was true. Even though the blood picture improved under the use of specific preparations (liver extracts, etc.), the damage to the liver seemed permanent, as evidenced by the galactose test. Patients with obstructive icterus show the same picture. The galactose test proved negative in a case of syphilis of the liver.

Three determinations of the blood sugar generally suffice to give a fair picture of the function of the liver.

SCHLUTZ, Chicago.

SCHIZOSACCHAROMYCES HOMINIS, BENEDEK, 1927: A PERMANENT ENDOPARASITE OF HUMAN BEINGS IN THE BLOOD OF THE UMBILICAL CORD. T. BENEDEK, Ztschr. f. Geburtsh. u. Gynäk. **104**:119, 1932.

The author believes that Schizosaccharomyces hominis is a human endoparasite the study of which may lead to the solution of many problems. It can be demonstrated in pure culture in approximately 90 per cent of blisters caused by cantharides. It has been demonstrated that this organism is really a human endoparasite.

There is intra-uterine transmission of the infection caused by Schizosaccharomyces hominis from mother to fetus. The organism can be demonstrated by the centrifugation of blood from the umbilical cord and the making of microscopic preparations. It can also be shown by culturing blood which is obtained from the umbilical cord under strict aseptic precautions.

Six hundred and fifty-seven specimens of umbilical blood were examined culturally in two separate series run at different times. There were 251 cases in the first series, with cultural demonstration in about 55 per cent. The second series consisted of 406 cases; the presence of the bacillus was shown in about 17 per cent of the cases. This amounts to about 32 per cent in the combined series.

ADAIR, Chicago.

THE HEMATOLOGY OF CHILDHOOD: II. HEPATOMEGALY AND GASTRO-INTESTINAL BLEEDING. EUGEN STRANSKY, Ztschr. f. Kinderh. **53**:92, 1932.

Five cases of gastro-intestinal bleeding in children are presented. In one the bleeding was related to thrombosis of the splenic vein with subsequent splenomegaly; in another, to splenomegaly without venous thrombosis or stenosis, and in two to portal cirrhosis in early childhood; in the last case, the cause of the gastro-intestinal bleeding could not be determined. This case occurred in a young infant (still living) who vomited blood-stained material and passed blood in the stools. There was secondary anemia with an increased number of reticulocytes. The bleeding time, the coagulation time, the clot retraction time and the platelet count were all normal. The spleen and the liver were not enlarged. Abnormalities of the gastro-intestinal tract were not found. Later ataxia and other manifestations of cerebral injury developed which might be due to hemorrhage into the substance of the brain.

SHELLING, Baltimore.

ACUTE INFECTIOUS HEMOLYTIC ANEMIA (LEDERER). FELIX ALTMAN, Ztschr. f. Kinderh. **53**:112, 1932.

A case of infectious hemolytic anemia in a boy aged 13 is presented. Certain features of the case were those of pernicious anemia. The acute fever, the rapid destruction of blood and the favorable response to therapy differentiate this type of anemia from pernicious anemia and hemolytic icterus.

SHELLING, Baltimore.

ACID-BINDING CAPACITY OF NORMAL AND PATHOLOGIC SPINAL FLUIDS. E. BERGAU, Ztschr. f. Kinderh. **53**:118, 1932.

The acid-binding capacity of fifty specimens of spinal fluid, determined titrimetrically, varied with the Pandy reaction. The stronger the Pandy reaction (increased concentration of protein), the greater the acid-binding capacity of the fluids.

SHELLING, Baltimore.

CANCER OF THE FEMALE GENITALIA IN CHILDREN. K. K. ORTMANN, Ztschr. f. Krebsforsch. **37**:283, 1932.

The author reports a case of vaginal cancer in a child 1½ years of age. Morphologically, the cancer was of an undifferentiated type; the photomicrographs suggest a similarity to the less differentiated squamous cell carcinomas, but it is stated that there were occasional suggestions of tubule formation. Two other cases of vaginal cancer and six of uterine carcinoma occurring in children are cited.

EGGERS, Omaha. [ARCH. PATH.]

PHOSPHATIDE DEGENERATION OF THE CELLS OF THE BRAIN, LIVER AND SPLEEN IN NIEMANN-PICK'S DISEASE. EMIL EPSTEIN and KARL LORENZ, Ztschr. f. physiol. Chem. **211**:217, 1932.

The chemical examination of the brain, liver and spleen in a case of Niemann-Pick's disease showed that in the process of phosphatide degeneration of cells not only the unsaturated but also the saturated phosphatides participate to a considerable degree. The phosphatide content of these organs is from eight to ten times the normal. The liver is the organ most affected.

HUEPER, Philadelphia. [ARCH. PATH.]

EXPERIENCES IN THE TREATMENT OF JUVENILE DIABETES MELLITUS. FRANTISEK BLAZEK, Časop. lék. česk. **70**:1232 (Sept. 4); 1255 (Sept. 11) 1931.

The author discusses the marked improvement in the treatment of diabetic children since the advent of insulin and, especially, the improved facilities for growth and development opened up by the new regimen. A series of thirty-five patients, twenty-two boys and thirteen girls, ranging from 18 months to 14 years is the basis for this paper. The requirements in the diet, together with factor ratios, are discussed. Several cases are reported in detail, and charts are presented showing the results obtained. In one girl an area of atrophy of the areolar tissues developed above the elbows after repeated injections of insulin. Hospitalization is recommended even in mild cases, so that a complete work-up can be made and a definite regimen instituted. Insulin is essential for carbohydrate equilibrium in practically all cases. The amount of insulin is usually progressive as weight increases and must usually be increased during intercurrent infection. Children apparently develop normally when a maintenance diet is covered with sufficient insulin to effect carbohydrate equilibrium. Apparently insulin has no effect on cataract, but in one case a diabetic xanthoma disappeared. Rotation of the sites of injection should be employed.

STULIK, Chicago.

DERIVATIVES OF CAFFEINE IN THE TREATMENT OF ASTHMATIC BRONCHITIS. C. W. HERLITZ, Acta pædiat. **13**:203 (June 6) 1932.

For the relief of bronchial spasm, epinephrine and atropine have the disadvantage that they must be given by injection. For oral administration the author recommends tablets containing 15 mg. of theobromine calcium salicylate and 35 mg. of theophylline. A child may take as many of these tablets as his age in years divided by two. The taste may be masked with honey.

McINTOSH, New York.

THREE SISTERS WITH DIABETES. U. HJÄRNE, Acta pædiat. **13**:225 (June 6) 1932.

The parents and an older brother showed normal dextrose tolerance curves. No family history of diabetes could be traced. In the three sisters, symptoms of diabetes appeared within thirteen months, when the patients were 3, 4 and 7 years old, respectively.

McINTOSH, New York.

EXCRETION OF CITRIC ACID THROUGH THE KIDNEYS IN CASES OF PYLOROSPASM. H. MAGNUSSON, Acta pædiat. **13**:345 (June 6) 1932.

As vomiting was brought under control by nonoperative measures, the urinary excretion of citric acid as determined by Thunberg's method fell in twenty-four hours from 121 mg. to 50 or 60 mg. In other conditions the output of citric acid has been shown to vary with the carbon dioxide-combining power of the plasma. While the carbon dioxide-combining power was not determined for Magnusson's patient, the author infers that the alkalosis of repeated vomiting was responsible for the increased output of citric acid.

McINTOSH, New York.

INFANTILE GLYCOSURIA SIMULATING DIABETES. B. STRANDQVIST, Acta pædiat. **13**:421 (June 6) 1932.

A subcutaneous abscess developed on the right shoulder of an infant, aged 1½ months. The urine contained 8 per cent dextrose but no ketone bodies; the blood sugar was 400 mg. per hundred cubic centimeters. The surgical condition was treated, and the infant was fed breast milk and was given insulin up to 2 units a day for two weeks. The disturbance in the regulation of blood sugar disappeared in about a month, and the abscess healed. Subsequently the patient's progress was normal. Strandqvist interprets the condition as a temporary dysfunction resembling, but not to be mistaken for, diabetes mellitus.

McINTOSH, New York.

Nervous Diseases

SELF-MUTILATION IN CHRONIC ENCEPHALITIS: AVULSION OF BOTH EYEBALLS AND EXTRACTION OF TEETH. S. P. GOODHART and NATHAN SAVITSKY, Am. J. M. Sc. **185**:674 (May) 1933.

The case of a Russian-Jewish girl, aged 16 years, with chronic encephalitis is reported. At 13 years of age the girl would occasionally extract her own teeth. While under observation in a general hospital about two or three years later, the patient gouged out her eyes. A review of the literature in which rare instances of this condition are reported is presented.

HENSKE, Omaha.

STUDIES OF CEREBRAL FUNCTION IN LEARNING: X. THE EFFECTS OF DILATATION OF THE VENTRICLES UPON MAZE-LEARNING. K. S. LASHLEY, W. T. McDONALD and H. N. PETERS, Am. J. Physiol. **104**:51 (April) 1933.

The effects of dilatation of the lateral and third ventricles of the brain on the rat's ability to learn mazes have been tested. Artificial enlargement of the

ventricles to as much as sixty times their normal volume by the injection of oil is followed by only a slight retardation in learning, which is scarcely greater than would be expected from the damage to the cortex by the injecting needle. The records of twenty-two animals with spontaneous hydrocephalus are not significantly different from those of others having equal amounts of cortical destruction and no hydrocephalus. The evidence rules out trophic disturbances arising from the mechanical distortion of tissue or increased intracranial pressure as a factor in producing correlation between the extent of cerebral destruction and efficiency in learning.

NOURSE, Cleveland.

CLINICAL CONVULSIONS. FOSTER KENNEDY, Am. J. Psychiat. **11**:601 (Jan.) 1932.

Epileptic seizures constitute no disease unit, but must be considered rather the expression of an instability of nerve centers of diverse origin operating through a common connecting mechanism, probably the cardiovascular system. The tonic phase may best be understood as a sudden cutting out of the highest level, so that the cortex permits the lower neuronic strata to pour downward an ungoverned stream of tonic postural impulses. The gradual return of cortical influences, incoordinate and involitional, produces the clonic phase. Of similar pattern, but of different intensity, are all epileptic phenomena from the seizure of grand mal to internally produced periodic emotional storms. The olfactory, auditory and visual hallucinations and illusions which often precede, accompany or replace epileptic seizures are explicable on a phylogenetic basis. Each person in his ascent from embryonic life traverses the stages through which the race has passed, and these sensory apparitions may be considered involuntary resuscitations of early and infantile memory patterns.

Of all extrinsic factors that render a brain unstable, trauma is one of the commonest. In 4 per cent of the cases of unstable brain occurring during the war, convulsions followed injuries to the skull and the brain; a dose of standardized convulsant too small to explode an intact brain will explode an injured one. The nature of the seizure depends on the location of the sensitive area; petit mal is associated with disorder of the frontal lobe, myoclonic attacks with striatal implication, cataplexies with thalamic involvement and hallucinations with pathologic conditions of the appropriate sensory cortices, while the grand mal is to be considered a sudden cutting out of cortical totality.

Many patients suffering from periodic convulsive disorders present no evidence of external or intrinsic cerebral trauma; in some of these an unlooked for trauma at birth may prove to be the predisposing agency. Bloody spinal fluid has been found in one hundred cases of apparently normal delivery, while in delivery with forceps, in breech delivery and in prolonged labors the percentage of cerebrospinal hemorrhage is even higher. This suggests an approach to preventive medicine in the field of epilepsy requiring the cooperation of the obstetrician. Once the injury has been accomplished, efforts must be directed, first, toward raising the threshold of neural explosion, and second, toward abolishing all possible detonating stimuli. Starvation, removal of infectious foci and the ketogenic diet are directed toward the latter goal, while the use of sedatives, notably phenobarbital, is at the time the best available method of raising the neural threshold.

DAVIDSON, Newark, N. J. [ARCH. NEUROL. & PSYCHIAT.]

MENTAL DETERIORATION AND DEFICIENCY: ITS CONSIDERATION FROM THE STANDPOINT OF CERTAIN CEREBRAL VASCULAR VOLUME RELATIONSHIPS. TEMPLE FAY, Am. J. Psychiat. **12**:893 (March) 1933.

Five groups of cases are discussed in which mental deterioration, acute or chronic, was due to intracranial spinal fluid pressure: group I, tumors of the brain associated with acute pressure; group II, acute communicating hydrocephalus due

to inflammatory obstruction of the basal cistern or sylvian sulcus; group III, acute hydration states associated with rapid mental failure; groups IV and V, posttraumatic neurosis and epilepsy, respectively. In all of these cases, the dehydration treatment (hypertonic solutions and limitation of fluids) and repeated spinal punctures proved valuable in relieving pressure symptoms, and in cases in which the causative factor was eliminated, this treatment brought about permanent relief from the mental disturbance. A diet for maintaining dehydration is appended.

JAHR, Omaha.

CONVULSIVE DISORDERS OF TWO OPPOSITE PERIODS OF LIFE, PUBERTY AND CLIMACTERIUM. ALFRED GORDON, Am. J. Psychiat. **12**:929 (March) 1933.

In view of the fact that many persons have their first epileptic seizure at the time of puberty and a cessation of these attacks at the time of the involutional period of life, the author asks, "Is the chronological relationship between the genital life and convulsive seizures a mere coincidence or is it fundamentally physiological?" He states that incidental coincidence, in the face of so many observations, must be discarded. Cases are cited in which epileptic fits were synchronous with menstruation and ceased during pregnancy and lactation, recurring with the resumption of menstrual regularity. While not all epileptic patients behave so typically, the author thinks that there may exist a sexual factor common to both ovulation and epileptic seizures. This factor is probably a noxious substance or a metabolite resulting from endocrine action.

JAHR, Omaha.

EPHEDRINE IN THE TREATMENT OF NARCOLEPSY. HARRY A. COLLINS, Ann. Int. Med. **5**:1289 (April) 1932.

The literature is reviewed, and the disease described. Somnolence and cataplexy coexist in this disease. It is idiopathic. The etiologic factors are unknown, and it cannot be explained on a functional or a hysterical basis. There is evidence to suggest that it is the result of a lesion in the region of the posterior portion of the third ventricle. Cases have been reported as early as the eighth year of life. It appears to occur more frequently in males (Cave gives the proportion of its incidence in males and females as 4:1). The disease is essentially chronic, and the duration is unknown.

Two cases are reported, one in a man, aged 34, and the other in a boy, aged 13. Ephedrine was given in doses of from 0.025 to 0.05 Gm. three times a day with beneficial results. Time will be required to evaluate the effect of ephedrine properly.

WILSON, Norfolk, Va.

THE VEGETATIVE NERVOUS SYSTEM IN CHILDREN WITH HEART DISEASE. MARTIN M. MALINER, Arch. Pediat. **50**:130 (Feb.) 1933.

Fifty children with chronic cardiac disease were given a subcutaneous injection of a 1:1,000 solution of epinephrine hydrochloride and the following conclusions were arrived at: 1. There are three distinct types of systolic blood pressure curves, suggesting a predominance of vagotonia or sympatheticotonia. 2. Cases in which the condition becomes progressively worse are all vagotonic. 3. The majority (64 per cent) of the cases are vagotonic. 4. Cases in which the condition is mild and improving are sympatheticotonic. 5. There seems to be a reciprocal relation between the vegetative nervous system and heart disease in children.

WADDELL, JR., University, Va.

THE TIME OF APPEARANCE OF EPILEPTIC SEIZURES IN RELATION TO AGE, DURATION, AND TYPE OF THE SYNDROME. HELAN HOPKINS, J. Nerv. & Ment. Dis. **77**:153 (Feb.) 1933.

In the epileptic patient the time of seizures tends to conform to a definite pattern. This paper deals with an analysis of 302 cases of the epileptic syndrome, 65 of which

were considered to be early cases of a duration of one year or less. The patients were noninstitutional, many self-supporting and others attending higher educational institutions. There were 209 adults and 93 children under 15 years of age. In 155 cases the condition was considered idiopathic, and in 147 organic. These cases could be definitely divided into diurnal and nocturnal with regard to the time of the seizures.

The largest number of attacks occurred during the sleeping state. Seizures tend to occur within narrower limits of time in younger patients, and as age increases there is a spreading out of the attacks to the diurnal or awakening period. The same tendency is also observed in the progression of the disease. No special difference in the time relationship was observed between the idiopathic and the organic type. The younger patients generally had their attacks over a more limited period of time than the older patients; moreover, the width of the range increased with the duration of the disease. In cases of a duration of a year or less, the largest number of patients had their attacks at 4 a. m.; in cases of from two to fifteen years' duration the time peak was reached at 6 a. m. A study is now in progress to determine the chemical factors precipitating the convulsions.

JAHR, Omaha.

EXPERIMENTAL CONVULSIVE SEIZURES. S. BERNARD WORTIS, J. Nerv. & Ment. Dis. **77**:233 (March) 1933.

Generalized convulsions in the cat were induced by injecting intravenously a solution of monobromated camphor. The following results were noted: generalized myoclonic convulsive discharges, fleeting pupillary dilatation with occasional enuresis and defecation, increased cardiac and respiratory rates, an increase in the deep reflexes and sweating of the pads of the feet. With the minimal convulsive dose there was blanching of the mucous membranes associated with momentary hypertonicity and gradual development of a generalized convulsive state (clonic). This stage was followed by loss of reflex excitability with its attendant relaxation, simulating symptomatically the epileptic syndrome.

The author gives a comprehensive discussion of the history and results of experimental convulsions produced by stimulating the various portions of the brain (surgically), together with a physiologic analysis of the seizures produced.

JAHR, Omaha.

DELAYED MENTAL DISORDERS FOLLOWING CRANIAL TRAUMATISM AND THEIR PSYCHO-PATHOLOGICAL INTERPRETATION. ALFRED GORDON, J. Nerv. & Ment. Dis. **77**:259 (March) 1933.

The cases of eleven patients with a history of cranial injury who later suffered from mental disorders were analyzed. In some patients psychotic symptoms developed many months after the injury. In all of the patients, the author concludes, "one could see deeply seated trends of emotional character dating from infancy ready to become exteriorized in an abnormal way under the influence of some affective stimuli." In each case there was a period of unconsciousness of from ten hours to five days, with absence of focal symptoms. Yet one cannot escape the fact that while no gross pathologic process is evident in such cases, changes of a diffuse character do take place (small hemorrhages in the brain and meninges, disturbances in the blood vessels and in the secretion of spinal fluid, changes in the choroid plexus and other changes). One cannot but conclude that the trauma serves merely as the exciting cause in the production of the disturbed mental state.

JAHR, Omaha.

RELATION OF JUGULAR FORAMINA TO EPILEPTIC SEIZURES AND HYDROCEPHALUS. D. B. DAVIS, J. Nerv. & Ment. Dis. **77**:351 (April) 1933.

Recent developments in the study of epilepsy indicate that developmental anomalies are an operative cause in about 20 per cent of cases. This report deals

with a group of fifty-two patients between 3 and 54 years of age. Measurements in square millimeters were taken of the jugular foramina by means of x-ray films, a special angle technic being used. Twelve plates were entirely discarded because of a defective outline of the foramina which made measurement impossible. In 72.5 per cent of forty patients (epileptic) the foramen on the right was larger, and in 27.5 per cent that on the left. In nine of ten patients with various conditions, the foramen on the right was larger, and in one, that on the left. In six of seven patients with hydrocephalus the foramina were too minute for accurate measurements.

JAHR, Omaha.

THE HEEL TAP, A PATHOLOGICAL REFLEX. SAMUEL M. WEINGROW, J. Nerv. & Ment. Dis. **77**:492 (May) 1933.

The reaction consists of tapping the heel of the foot while the foot is held in any position comfortable to the examiner and the patient. A positive reaction is characterized by fanning, and in many cases by plantar flexion of the toes. Any movement of the toes following the heel tap may be considered pathologic. More than 200 normal persons showed no movement of the toes following tapping of the heel. A positive reaction indicates advanced involvement of the pyramidal tract.

JAHR, Omaha.

ACUTE POLIOMYELITIS: STUDIES OF STREPTOCOCCI ISOLATED FROM THROATS AND RAW MILK IN RELATION TO ONE EPIDEMIC. EDWARD C. ROSENOW, HENDRIK M. ROZEMDAAL and E. T. THORSNESS, J. Pediat. **2**:568 (May) 1933.

An epidemiologic and experimental study of an epidemic of poliomyelitis due to raw milk is reported. By means of the precipitin reaction between cleared extracts of nasopharyngeal swabbings and the poliomyelitic antistreptococcus serum, the incidence of carriers was quickly determined. The presumed susceptibility to poliomyelitis was tested on all school children by cutaneous tests with poliomyelitic streptococcus antigen. The streptococcus was isolated from eight brands of raw milk supplied to families in which fourteen cases of poliomyelitis occurred. The streptococcus was also found in several samples of pasteurized milk. Success in isolating the streptococci from the milk is attributed to cataphoretic studies and to the use of dextrose-brain broth as a culture medium. The authors state that there are a number of facts which indicate that the neurotropic velocity of the streptococci in the raw milk at the time of the epidemic represents a temporary, acquired property of green-producing streptococci commonly present in the milk of healthy cows. Adequate pasteurization and refrigeration are necessary to make the milk safe for consumption. The streptococcus isolated from the raw milk at the time of the epidemic was considered to be identical with that isolated in cases of poliomyelitis occurring in human beings and in monkeys in its peculiar virulence, its power to cause flaccid paralysis in rabbits, its thermal death point, its morphology and cultural characteristics, its immunologic properties and its cataphoretic velocity.

LEMASTER, Evanston, Ill.

PSYCHOLOGICAL MALADJUSTMENT IN AN ADULT DUE TO PHYSICAL DEFORMITIES CONTRACTED IN CHILDHOOD. GEORGE P. REYNOLDS, M. Clin. North America **16**:1011 (Jan.) 1933.

Reynolds reports a case in order to illustrate the profound psychologic disturbance which may result from maladjustment to physical deformities in childhood. A boy who was crippled by poliomyelitis at 2 years of age was not recognized as having any abnormal psychologic reaction until he had grown to manhood. At the age of 23 years, he was uncouth in appearance, practically friendless and desperately lonely. The treatment consisted simply in an explanation of his abnormal reactions. This case represents a type in which preventive psychotherapy may be most successfully applied.

EUGENE SMITH, Ogden, Utah.

REPORT OF THE COMMITTEE ON MENTAL DEFICIENCY. PRESENTED TO THE ANNUAL REPRESENTATIVE MEETING OF THE BRITISH MEDICAL ASSOCIATION, JULY, 1932.

The terms "idiot," "imbecile" and "feeble-minded," as defined by the British Mental Deficiency Act, are acceptable to the committee, although the expression "moral defective" meets with less universal approval. Excluded from the category of moral defectives are delinquents whose difficulties are due in large part to social and emotional maladjustments, and persons suffering from epilepsy, syphilis, encephalitis, injury to the head, chorea and the neuroses, whose inadequacies can be explained on the basis of the disease picture. However, "moral defect" is properly included within the broad scope of mental deficiency on the theory that it is due to a failure of the subject to develop the capacity for wisdom, judgment and control, based possibly on actual cerebral disease. Morally defective persons, because of their normal intelligence and persistent tendency toward delinquency, are not amenable to ordinary institutional care and need special accommodations.

The committee is satisfied that heredity is an influence in the determination of mental deficiency, but is unable to state in what proportion of cases it constitutes a significant factor. It seems likely that there are several congenital bases, the trend being transmitted as a multiple recessive complex of traits. The x-rays and radium can certainly produce inheritable mutations in the genes, but the influence of chemical toxins is less definite. There is no reason to believe that tuberculosis, lead poisoning or intoxication due to mercury in either or both parents is likely to be followed by defective offspring. Syphilis may certainly be associated with mental defect, but the relationship is not so simple and direct as is commonly assumed. The coexistence of clinical evidence of congenital syphilis, a positive Wassermann reaction and mental deficiency constitutes no proof that the mental condition is due to syphilis. In a definite but small number of cases, alcoholism plays a part. The claim that, in any particular case, injury at birth was the precipitating factor must be examined with critical care, for the sequence does occur but is rare. Postnatal trauma is an even more infrequent etiologic factor. Infections, especially encephalitis, seem in many instances to be the more likely agencies.

The committee warns against the stigmatizing of a child as mentally defective because of inadequate success at school, pointing out that failure at school is not incompatible with excellent social adjustment. It recommends the utilization of tests in achievement and aptitude as supplementary to the conventional psychometric examination before a diagnosis is made.

For the care of the mentally defective, the committee much prefers the large colony to the small home or the hospital for patients with mental conditions. The large colony possesses many advantages: Employment can be organized on a scale sufficiently wide to assure suitable work for every inmate; patients of higher grade mentality can be segregated and specially trained; economy is effected by reduction in the overhead charges, and life can more closely approximate normal communal intercourse.

The only method of prevention discussed is sterilization; the committee is uncertain as to the desirability of this, feeling that if the operation were universally applied to all of the commitable feebleminded, no reduction in the incidence of mental deficiency would be noted. This is because of the large numbers of unreachable carriers who could transmit the defect. Even if those patients whose chief social danger is propagation were sterilized, the advantages would be uncertain because of the sexual promiscuity and consequent spread of venereal disease to which such a practice would lead.

The committee is inclined to look for relative disproportion in the intra-uterine development of neuroblasts and spongioblasts as a common pathologic base for many cases of mental deficiency. Certainly cases of hydrocephalus and microcephalus can be explained in this manner. The controversy as to heredity and environment is resolved by the committee in the statement: "Heredity furnishes the material, environment shapes and uses it; heredity is the mechanism, environment,

the stimulus which sets it in action; heredity fixes the possibilities of development, environment determines which of these possibilities shall, and how far they shall, become realities." Studies in comparative anatomy and anthropology seem to suggest that it is to a defect in the number rather than in the structure of the brain cells that one should look for the factors that underlie mental deficiency.

Many of the maladjustments suffered by the feebleminded are explicable more on the basis of their environmental difficulties than on the theory of their fundamental inadequacy. The high grade moron who adequately performs a humble and necessary task is an effective unit in the social machinery and should not be patronized as "unfit." Certainly, antisocial conduct is not even a common, much less a necessary, characteristic of the mentally deficient. These people need to be protected from society as much as society needs to be protected from them. The committee considers that this rapidly growing problem is generally overstressed, for it has found no evidence that procreation is excessive among the feebleminded, or that the number of children in mentally deficient families who reach adolescence exceeds the number in normal families.

The report is concluded with several appendixes dealing with the problem of marriage, illegitimacy and community care of the mentally deficient. Emphasis is laid on the ultimate aim of the recommended program—the socialization of the mental defective. DAVIDSON, Newark, N. J. [ARCH. NEUROL. & PSYCHIAT.]

FACIAL PARALYSIS DUE TO TOXIC INFLAMMATION OF THE GENICULATE GANGLION. J. P. FINDLAY, M. J. Australia **1**:251 (Feb. 25) 1933.

Findlay reports five cases of facial paralysis due to toxic inflammation of the geniculate ganglion. The characteristic symptoms of the condition consist of intense otalgia; tinnitus; swelling of the ear, which develops into herpes zoster, and loss of taste. The herpes zoster involves the drum membrane, the walls of the external aural canal, the external meatus, the cavum conchae, the antitragus, the anthelix and part of the lobule. This inflammation is the result of a focus of infection in the mouth and pharynx. Treatment consists in eradication of the focus of infection, early massage and early electrical stimulation of the facial muscles.

GONCE, Madison, Wis.

CEREBRAL TUMOURS: EXPERIENCES AT THE ROYAL ALEXANDRA HOSPITAL FOR CHILDREN. F. TIDSWELL, M. J. Australia **1**:280 (March 4) 1933.

Tidswell gives an analysis of twenty-two tumors of the brain in respect to pathologic types, symptoms and signs. Ten of the tumors were cerebellar astrocytomas. The author makes a plea for early diagnosis.

GONCE, Madison, Wis.

ACUTE POLIOMYELITIS. D. STEWART MIDDLETON, Practitioner **130**:487 (April) 1933.

The early history of acute poliomyelitis is reviewed. It has been shown to be an infectious fever and to belong to the class of diseases due to a virus.

In monkeys, experimentally, the period of incubation varied from three to thirty days. The period is still in doubt, owing to lack of data. The author describes six types of poliomyelitis: (1) the spinal, (2) the meningeal, (3) the progressive ascending, (4) the bulbar, (5) the polioencephalitic and (6) the ataxic.

As to diagnosis, Middleton states that there is no definite symptom complex which will make a diagnosis in the preparalytic stage anything more than an intelligent guess. He advises a spinal puncture in suggestive cases and during epidemics.

The early pathologic condition consists in a marked inflammatory reaction and edema. The latter affects mainly the anterior horn cells of the cord and may involve the posterior root and the sensory ganglion.

With regard to treatment, the author feels that during the stage of invasion the use of immune serum is indicated. At present it is impossible to estimate the value. When immune convalescent serum is not available, serum from a healthy adult should be given.

The importance of complete rest during the paralytic stage, the proper splinting of muscles, the reeducation of muscles during the stage of recovery and the correction of deformities are given careful consideration.

SAPPINGTON, San Francisco.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS—A CASE WITH ASYMMETRICAL ATROPHY. W. G. WYLLIE, Proc. Roy. Soc. Med. **26**:284 (Jan.) 1933.

A boy of 8 years had appeared to be healthy and to develop normally until the age of 7 years, when it was noticed that the right arm was thinner and weaker than the left. All the muscles of the right shoulder and arm were weak, and there was considerable wasting of the trapezius, serratus magnus, latissimus dorsi, supraspinatus and infraspinatus, rhomboid, deltoid, triceps and biceps muscles. On movement, but not during rest, there was a twitching resembling fibrillation in the muscles of the upper arms. In the left arm the only abnormal sign was early winging of the scapula. The muscles of the calves of the legs were prominent and tough, but both knee and ankle jerks were obtainable. The parents were not related, and the patient's only brother was healthy.

WILLIAMSON, New Orleans.

ENCEPHALITIS PERIAXIALIS (SCHILDER). E. D. MACNAMARA, Proc. Roy. Soc. Med. **26**:297 (Jan.) 1933.

The author reports a case of encephalitis periaxialis in a girl of 5½ years. An elder sister was suffering from the same disease, and two other sisters had died of it. Full notes on the progress are given in the report, as well as pictures of many sections of the brain taken from other members of the patient's family.

WILLIAMSON, New Orleans.

TACCONE'S REACTION IN THE CEREBROSPINAL FLUID. A. PACIFICO, Riv. di pat. nerv. **40**:1 (July-Aug.) 1932.

The author has investigated Taccone's reaction in the cerebrospinal fluid by comparing the reaction in various non-neurologic and various neuropsychiatric conditions. The reaction is produced by adding to a 5 per cent solution of potassium bichromate to which trichloracetic acid has been added 0.5 cc. of cerebrospinal fluid, which is layered on with the reagent. At the point of separation of the fluids a whitish ring is formed, owing to precipitation of the proteins by the trichloracetic acid. If the ring remains homogeneous and disappears gradually within four or five hours, the reaction is considered negative. In a positive reaction the ring may assume the aspect of a spider's web, which fluctuates in a direction parallel to the major axis of the test tube, and which precipitates to the bottom of the tube within the first three hours. At other times the ring remains adherent for several hours to the walls of the test tube. Again, a more or less dense coagulum, irregular in its margin, is formed and gradually reaches the bottom of the test tube. The reading should be made between the second and third hour after the contact of the reagent with the cerebrospinal fluid.

With the technic described, Pacifico investigated more than 150 specimens of cerebrospinal fluid; he arrived at the following conclusions: 1. The Taccone reaction is technically simple, the reagents are stable, reading is easy and there is marked sensitiveness. 2. The determination of a positive reaction is probably not influenced by the presence in the fluid of oxidizing substances or ferments, but is related to an increase in protein substances in general. The action of the bichromate is limited presumably to hardening of the proteins which are

precipitated by the trichloracetic acid. 3. The appearance of a positive reaction (fluctuating ring) and of the so-called encephalitic reaction (adherent ring) in cerebrospinal fluid containing normal protein discloses a slight increase of globulin (as low as 6 or 8 cg.). This slight change which the Taccone reaction reveals is below the threshold of sensitivity for other common tests for globulin (Pandy, Nonne-Apelt and Weichbrodt reactions).

The sensitivity of the reaction makes it particularly available in furnishing the first indication of an initial biochemical modification of the cerebrospinal fluid and therefore constitutes an element of value for diagnosis and prognosis in indicating the existence of an organic condition. The reaction is not characteristic of any particular disease and is helpful only in establishing the existence of involvement of the nervous system.

FERRARO, New York. [ARCH. NEUROL. & PSYCHIAT.]

THE CLAUDE BERNARD-HORNER SYNDROME AND INFANTILE CEREBRAL HEMIPLEGIA OF OBSTETRIC ORIGIN. T. R. YANES, Rev. cubana de oto-neuro-oftal. **1**:181 (May-June) 1932.

A white child, aged 12 years, with nothing of importance in the hereditary or familial history, presented a typical Claude Bernard-Horner syndrome on the left side, together with a shortening of the left arm and leg, equinus deformity and ankylosis of the left foot, spastic gait and muscular atrophy of the entire left side of the body.

The infant was born at eight months, after the forceps had been applied five times; it was cyanotic, showed two wounds in the right parietotemporal region, and in six hours exhibited an enormous tumefaction of the neck and head which lasted twelve hours.

The atrophic phenomena began to manifest themselves about the age of 13 months; the ocular findings were apparently the same as at the time of birth. The later findings were: ptosis of the superior lid of the left eye with narrowing of the palpebral fissure, enophthalmos, anisocoria with left myosis, heterochromia, convergent strabismus and left facial atrophy.

Regarding the cause, the author feels that possibly the blade of the forceps over the right temporal region produced an intracranial hemorrhage in this area, while the blade over the left cervical region caused injury of the peripheral sympathetic nerve. Added to this, there may have been trauma to the sympathetic centers of the bulb.

RHODES, Reading, Pa. [ARCH. OPHTH.]

ARACHNITIS ADHESIVA CIRCUMSCRIPTA IN A PATIENT WITH LATE RACHITIC DEFORMITY OF THE VERTEBRAL COLUMN. R. GRÜN, Ztschr. f. d. ges. Neurol. u. Psychiat. **138**:428, 1932.

Arachnitis has been described as a secondary disorder in a number of conditions —acute and chronic infections, inflammations, tumors and injuries of the spinal column, the spinal cord and its membranes and infection of the nervous system. In addition to these there is a group of cases of unknown etiology, spoken of as the primary form of arachnitis. Grün reports a case of this type which occurred in a patient with late rachitic deformity of the vertebral column.

ALPERS, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

CHANGES IN THE CORNU AMMONIS IN AMAUROTIC FAMILY IDIOCY. HANS JOACHIM SCHERER, Ztschr. f. d. ges. Neurol. u. Psychiat. **138**:481, 1932.

Amaurotic family idiocy, a ubiquitous nerve process, tends sometimes to be accentuated in certain areas. Bielschowsky found it outspoken in the cerebellum in some cases, and Meyer in the basal ganglions. The accentuation of the process in the cerebrum has not been recorded. Zierl mentioned that the cornu ammonis was mostly involved in a case studied by him, and Kiefs made the same assertion in a case recently reported.

Scherer studied the cornu ammonis in ten cases of amaurotic idiocy. In all these the process was definitely accentuated. The distribution of the involvement is of interest. Usually Sommer's sector suffers most in amaurosis, while the "resistive" portion of the cornu ammonis suffers least. In amaurotic idiocy, on the other hand, the most severe changes are found in the dorsal and terminal leaves, while Sommer's sector is resistive to the cell changes. The fascia dentata and subiculum are affected next to the dorsal leaf. The most vulnerable parts, therefore, are those whose cells are normally lipophilic.

ALPERS, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

ATHETOSIS BILATERALIS. H. A. STHEEMAN, Maandschr. v. kindergeneesk. **2**:318 (April) 1933.

The author describes a case of athetosis bilateralis observed in a boy of 7 years.

VAN CREVELD, Amsterdam, The Netherlands.

SIGNIFICANCE OF CHANGES IN THE VESSELS IN SUBACUTE PURULENT MENINGITIS. A. BIEMOND and J. GOUDSMIT, Nederl. tijdschr. v. geneesk. **76**:4200 (Sept. 3) 1932.

In a girl, aged 16, suffering from subacute purulent meningitis with a changing clinical course, neurologic symptoms on one side and an extension of the exudate mainly on one side, there was found post mortem marked endarteritis of the pial vessels as well as a degenerative change of the ganglion cells of the deeper layers of the cerebral cortex. This degeneration, which was apparently the cause of the neurologic symptoms, is regarded as a consequence of the ischemic degeneration resulting from the abnormality of the pial vessels. The authors suggest that this mechanism may play a rôle in many cases of subacute and chronic meningitis.

VAN CREVELD, Amsterdam, The Netherlands.

TWINS WITH EPILEPSY. J. SANDERS, Nederl. tijdschr. v. geneesk. **76**:4513 (Sept. 24) 1932.

The author reports the results of thorough studies with the newer methods of three identical twins and one nonidentical twin who had epilepsy.

VAN CREVELD, Amsterdam, The Netherlands.

THE NEUROPATHIC CHILD. P. TILMA, Nederl. tijdschr. v. geneesk. **76**:5759 (Dec. 17) 1932.

The neuropathic symptoms resulting from a faulty attitude of the parents toward the child are discussed. Too few children is often the cause of this fault, because the natural instincts of the mother are working in a false direction. The symptoms are divided into three groups: (1) those caused by forcing the child out of its natural path of development, (2) those caused by a too early development of self-consciousness, resulting in prematurity and a derangement of spiritual functions by perpetual interference, leading to a varied multitude of somatic complaints, and (3) derangement of the autonomic nervous system. Brief allusions are made to the principles of treatment.

VAN CREVELD, Amsterdam, The Netherlands.

AMAUROTIC FAMILY IDIOCY. K. T. A. HALBERTSMA and P. P. LEENDERTZ, Nederl. tijdschr. v. geneesk. **77**:377 (Jan. 28) 1933.

The author describes amaurotic family idiocy in two children of one family; a girl, aged 17, showed all the typical symptoms of the disease, whereas a boy, who was younger, showed only the typical deviations of the eyes. The significance of the deviations observed in the fundi of the healthy members of the family also in the distribution and hereditary transmission of the disease is discussed. In

agreement with the present conceptions, the condition in this case was a recessive abnormality which was made manifest by a consanguineous marriage. The conceptions of the disease and its cause are summarized.

VAN CREVELD, Amsterdam, The Netherlands.

DIFFUSE SCLEROSIS IN CHILDREN. L. BOUMAN, Nederl. tijdschr. v. geneesk. **77**:491 (Feb. 4) 1933.

The clinical picture of diffuse sclerosis is given. The author observed six cases and collected from the literature reports of one hundred cases, in fifty of which the patients were below the age of 15. The most important aids to diagnosis have been the results of autopsies. The clinical manifestations are extremely variable, but as the author shows, some combinations of clinical symptoms occur so commonly in this disease that probably in the future the diagnosis can more frequently be made during life.

VAN CREVELD, Amsterdam, The Netherlands.

Diseases of the Skin

HEMANGIOMA: SOME OBSERVATIONS ON THE RESULTS OF RADIATION THERAPY. HUGO ROESLER, Am. J. Roentgenol. **27**:249 (Feb.) 1932.

The author reports the cases of forty patients treated by various methods and watched for periods up to seven years. Twenty-five of the patients were under 2 years of age and thirty-four were under 20 years. The location of the hemangiomas was as follows: eyelids, fourteen; lips, four; nose, four; face and neck, sixteen; scalp, two, and other parts of body, four. In four cases the lesions were present on two different parts of the body. The treatment consisted of the application of radium in almost all the cases. The possible injuries are permanent epilation of the lashes and disturbance of tissue growth with scarlike shrinking of the lip or eyelid. The results were very satisfactory, especially in infants. The smallest dose with which an improvement was noted was one-half erythema dose. Superficial and smaller lesions respond to radium applied in any form. The standard treatment consisted of applications of steel needles at a distance of 1 cm., filtered through 1.5 mm. of brass, the dose of from 250 to 300 mg. hours corresponding to one-half erythema dose. In a majority of the cases from one-half to two-thirds erythema dose was required. This was repeated at varying intervals. The time between treatments should be as long as possible because improvement may be slow. Good results are at times obtained with roentgen rays, two-thirds erythema dose being used at one sitting. Large deep-seated tumors respond well, and ulcerated hemangiomas present no contraindications. The smallest possible dose which will bring results should be used because of better cosmetic results and because of the uncertainty of future tissue reaction.

STULIK, Chicago.

MULTIPLE FIBROMAS: RECKLINGHAUSEN'S DISEASE. JAMES T. WAYSON, Arch. Dermat. & Syph. **27**:421 (March) 1933.

A Chinese woman came to Hawaii at the age of 20. At that time her skin was without blemish. Shortly after her arrival, a few small tumors appeared. These increased in size and number until thirty years later she had thousands of fibromas. There were hundreds on the scalp and face, and many on the lips. The palms and soles and the interdigital spaces of all four extremities were covered with lesions. The patient had four children; the two sons and one of the daughters had numerous lesions. The other daughter, although free from tumors, had a goiter. A child born to the daughter with the fibromas when 9 months of age had large fontanels and was unable to raise its head. The author does not state whether a biopsy was done and what the pathologic study, if any was made, showed.

DAVIDSON, Newark, N. J. [ARCH. NEUROL. & PSYCHIAT.]

LYCOPODIUM GRANULOMA. WILLIAM ANTOPOL, Arch. Path. **16**:326 (Sept.) 1933.

In six patients who had been operated on a few weeks previously, chronic sinuses occurred. Microscopic sections from the walls of the tracts showed within the granulation tissue epithelioid cells and giant cells containing spores of Lycopodium. It was thought that the spores were introduced into the wound from the dusting powder used on the surgeon's gloves. The lesions caused by Lycopodium grossly resembled tuberculosis or neoplasm. In one instance the histologic examination of a grossly normal appendix revealed refractile bodies morphologically identical with crystals of talc. Since this patient had undergone two surgical operations eight months and eight years previously, it was considered likely that talc from the surgeon's gloves had been introduced at one of these times into the wall of the appendix when it was handled and inspected.

LEMASTER, Evanston, Ill.

SOY BEAN (VEGETABLE) MILK IN THE DIETETIC TREATMENT OF INFANTILE ECZEMA. FRED KITTINGER and LEAR H. DEMBO, M. J. & Rec. **138**:52 (July 19) 1933.

The author gives a list of articles showing various opinions of the etiology and treatment of eczema. He then gives the results of his own treatment by feeding soy bean milk. He gives the following summary of his work: 1. Twenty patients with infantile eczema were treated by the substitution of soy bean milk feedings for foods containing animal protein. 2. The average time of clearance of the lesions was six weeks, with a range from one week to three months. 3. There were recurrences in several cases when cow's milk and cereals were given. 4. The nutritional welfare of these babies was good, most of them gaining well. 5. There was complete clearance in fourteen of the twenty cases and partial clearance in six.

WILLIAMSON, New Orleans.

ECZEMA. CHARLES S. TOMLINSON, Nebraska M. J. **18**:343 (Sept.) 1933.

Diagnostic evolution has, in twenty-five years, reduced the frequency of diagnosis of eczema from 33 to 5 per cent. The cardinal characteristics of eczema can be recalled by the formula of the two "m's," standing for moisture and multiformity, and the three "i's," standing for indefinite outline, itching and infiltration. Eczema is produced by a great variety of causes. The author states that his experience has been that even though foods incriminated by scratch tests or by intradermal tests are eliminated from the diet, the eczema continues "in all its glory." Elimination diets are worthy of trial. It is recommended to start the patient on a diet restricted to those foods least likely to produce a reaction, then gradually adding to these and noting carefully any reactions. It is believed that the responsibility of a single food or foods has been greatly exaggerated. External irritants are not a negligible factor. A high percentage of all eruptions which satisfy the morphologic criteria of eczema are known to be caused by ringworm fungi. Proof of this may be established as follows: 1. The fungus cannot be recovered from the eczematous eruption, but can be recovered from the primary focus, which is usually on the feet. 2. The patient exhibits a positive reaction to intradermal allergic tests with an aqueous extract of ringworm fungi known as trichophytin. 3. The eczematous eruption involutes when the primary focus of ringworm infection is eradicated.

HAMILTON, Omaha.

ECZEMA: A SURVEY OF THE POSITION. J. FERGUSON SMITH, Glasgow M. J. **119**:145 (May) 1933.

Dermatitis in adults due to occupation and sensitization is discussed. The author advocates the use of the Bloch patch test. His impression is that some substance which early causes irritation of the skin, if continued, produces sensitization and chronic eczema develops. The Prausnitz-Küstner phenomenon, or passive

sensitization, should be used if one experiences difficulty in finding enough clear skin on which to make the tests or if there is a general hypersensitivity of the skin to any substance applied.

Eczema in infants is sensitization. "Irritation from any cause produces dermatitis, and if this is allowed to continue unchecked for, say a fortnight, 'eczema' follows." The primary cause in infants is microbic, which gives rise to the seborrhea starting on the scalp and cheeks. Scratching spreads the condition and permits sensitization. Very few cases are due to allergy to food protein. The author claims that diet plays a very small part in eczema in infants.

In treatment he advises prompt care for dermatitis, before it reaches the stage of eczema. Preparations of zinc oxide, of sulphonated bitumen and calamine and of crude coal tar are used locally. Roentgen therapy is used in the intractable cases, especially for the chronic hyperkeratotic eczemas of the hands and feet. Autohemotherapy, or the injection of 5 cc. of the patient's blood into the gluteal muscles, is used as a method of desensitization. Typhoid and Bacillus coli vaccine have been used, as well as methods to cause protein shock.

SNELLING, Toronto, Canada.

VITILIGO WITH GREY HAIR. JEAN SMITH, Proc. Roy. Soc. Med. **26**:1019 (June) 1933.

A case of vitiligo with gray hair in a girl of 8 years is reported.

WILLIAMSON, New Orleans.

GENERALIZED FAVUS. A. G. LOURIER and M. G. REIFF, Ann. de dermat. et syph. **3**:913 (Oct.) 1932.

The authors report a case of favus affecting the glabrous skin as well as the scalp of a girl, aged 14. Achorion schoenleinii was isolated by culture from both the blood and the lymph glands.

GANDY, Houston, Texas. [ARCH. DERMAT. & SYPH.]

ACNE VULGARIS AND CHRONIC ECZEMA CURED BY TONSILLECTOMY. A. GALTUNG, Norsk mag. f. lægevidensk. **94**:851 (Aug.) 1933.

The author describes three cases of acne vulgaris in which local treatment had given negative results. After tonsillectomy, however, the cutaneous lesions disappeared. The chronic tonsillitis in these patients had evidently caused the absorption of toxins and thus had produced an allergic condition of the skin.

TOVERUD, Oslo, Norway.

Miscellaneous

EFFECT OF A HIGH TROPICAL ALTITUDE ON INFANTS. C. TORRES-UMANA, Rev. franç. de pédiat. **9**:349, 1933.

The author writes from Bogota, Colombia. Conditions which are unfavorably affected in such an altitude (2,800 meters) are heart disease, renal conditions, arteriosclerosis and acidosis; those favorably affected are rickets, spasmophilia, scurvy, diseases due to tropical parasites, tuberculosis (the rate is 91 per hundred thousand persons) and contagious diseases. Diphtheria is very rare. The principal cause of mortality is gastro-intestinal disturbances (40 per cent).

SANFORD, Chicago.

HYGIENE OF THE NURSING INFANT. C. S. DE LOS TERREROS, Nipiologia **19**:11 (March) 1933.

The infant requires the same hygienic attention that the adult does. The room should contain an air space of 20 cubic meters. The temperature should be kept

at a constant mean between 34 and 37 C. (93.2 and 98.6 F.). The clothing should be warm and porous, but of such a quality and cut as to allow free muscular movement.

SANFORD, Chicago.

THE FIGHT AGAINST INFANT MORTALITY IN RURAL COMMUNITIES. LORENZO LOSTE ECHETO, Med. de los niños **33**:257 (Sept.) 1932.

In 22 European countries, including Rumania, Hungary, Czechoslovakia and Lithuania, the mortality in the first year of life increased in the biennium from 1927 to 1928. Deaths before the age of 1 year in the decennium from 1921 to 1930 were, per thousand, 70 in England, 60 in Holland, 60 in Switzerland, 91 in France, 124 in Italy and 133 in Spain. In the small towns the mortality is much greater, which the author attributes to ignorance on the part of the mother and the miserable living conditions. He recommends education of the mother in the superiority of breast feeding, the terrors of diarrhea, which is easier to prevent than to cure, and the need for vaccination, especially against smallpox and diphtheria.

DWYER, Kansas City, Mo.

PROF. A. SCHLOSSMANN. MARTINEZ VARGUS, Med. de los niños **33**:281 (Oct.) 1932.

The author laments the death of the great pediatrician who for thirty years taught at Dusseldorf and directed one of the greatest clinics in Germany. One of his greatest achievements was his textbook on pediatrics, in four volumes. The author was in charge of the translation of the work into Spanish and also collaborated on a review of pediatrics commemorating the sixtieth anniversary of Schlossmann's birth.

DWYER, Kansas City, Mo.

BLOOD TRANSFUSION IN THE INFANT. D. ENRIQUE ALBELDA VALLE, Med. de los niños **33**:296 (Oct.) 1932.

Transfusion of blood, used for a long time in adults, has been recently introduced in the therapy of children. Because of the technical difficulties, it is usually reserved for cases that are extremely grave, and because of this not much benefit results from the procedure.

The principal indications for transfusion in infants are disturbances of nutrition and diseases of the blood. The author discusses the value of large transfusions, i. e., 30 cc. of blood per kilogram, and small transfusions, from 20 to 30 cc., repeated frequently.

The technic used is that of dissection of a superficial vein and the introduction of a cannula, citrated blood being used. The most common disturbances are the result of too rapid injections, which cause disturbances of the circulation, such as cardiac dilatation, disturbances of rhythm, cyanosis and respiratory irregularity. These may be very alarming, but they are rarely serious. Inhalations of oxygen and the injection of camphor in oil and caffeine are used to stimulate the patient during a reaction.

DWYER, Kansas City, Mo.

DENTAL INFECTION AS AN ETIOLOGIC FACTOR IN CERTAIN DISEASES. BOY S. GARDNER, Med. de los niños **33**:322 (Nov.) 1932; Dent. Cosmos, May 1932.

The author discusses dental infections as a cause of disease of the eyes, blood, skin, joints, gastro-intestinal tract, urinary tract and heart and as a cause of abortion.

DWYER, Kansas City, Mo.

CARE AND TREATMENT OF THE TEETH IN CHILDHOOD. G. FISCHER, München. med. Wchnschr. **80**:30 (April 14) 1933.

Dental prophylaxis is most important and should be begun by the mother in the antepartum period. The diet during pregnancy should contain plenty of calcium

and vitamins. The child should begin to brush his teeth at 3 years of age. His diet should be well balanced and contain an abundance of calcium and vitamins. Infections injure the teeth, and therefore it is important to increase the child's resistance as much as possible.

BRAHDY, Mount Vernon, N. Y.

VALUE OF THE INSTITUTION IN THE CARE OF DEPENDENT CHILDREN. HANS NATHANSON, Ztschr. f. Kinderh. **41**:97 (Jan. 11) 1933.

This article represents a protest against the excessive use of the institution in the care of dependent children. The dangers to which a child is subjected when brought into a large institution with a mixed population are described. The final result of all social service appears to be that the best place for a child is a home with a small family.

REICHLE, Cleveland.

THE RELATION OF INTELLIGENCE TO SOCIAL POSITION. K. SALLER, Ztschr. f. Kinderforsch. **41**:181 (March 15) 1933.

These investigations were carried out in the schools of Hanover. The results are not new: Talented and mentally inferior children came from all social classes, but the percentage of those mentally inferior was higher among the working classes, whereas the talented children were found more frequently in the higher classes. The decrease in the birth rate is much less pronounced in the families with mentally inferior children than in the families with talented children.

REICHLE, Cleveland.

THE CHILD AND DEATH. ERICH STERN, Ztschr. f. Kinderforsch. **41**:221 (March 15) 1933.

The psychologist has investigated the attitude of children toward the subject of death and comes to the following conclusions: At the age of 10 years practically all children have encountered the idea of death. The subject is frequently regarded in a symbolic (dance of death) or in a superstitious fashion. Religious conceptions are meager. The child usually gives attention to the subject for the first time when there is a death in the family or in the immediate neighborhood. Not infrequently the thought that the child itself must die causes serious conditions of fear. It may even lead to the development of a neurosis. Frequently, however, one finds children 10 years of age who do not appreciate the full significance of death.

REICHLE, Cleveland.

CAUSES OF INEQUALITIES IN THE INFANT MORTALITY RATES OF VARIOUS COUNTRIES. J. A. HÖJER, Acta pædiat. **13**:238 (June 6) 1932.

The statistics for infant mortality in Sweden are available as far back as the middle of the eighteenth century. The curve fell slowly up to 1800, more rapidly throughout the nineteenth century and still more rapidly up to 1925, when the rate remained close to 60 for a few years. Sweden's method of combating infant mortality depended largely on the teaching of her medical students and on instruction of the lay public through printed propaganda.

In certain other countries in which the present century started with much higher infant mortality, the curve of the rates for successive years has fallen more precipitously than that of Sweden. The explanation lies in the adoption by these nations of active propaganda to carry instruction in health directly to the people in the form of clinics for antepartum care, clinics for healthy babies and demonstrations.

MCINTOSH, New York.

STUDY OF PEDIATRICS IN THE UNIVERSITY OF TARTU (1632-1932). A. LÜÜS, Acta pædiat. **13**:328 (June 6) 1932.

Lüüs presents a historical sketch of the teaching of pediatrics in an Esthonian university.

MCINTOSH, New York.

IMPORTANCE OF AEROPHAGIA FOR INFANTILE METEORISM. W. MAGNUSSON and H. ENGSTRÖM, Acta pædiat. **13**:348 (June 6) 1932.

Swallowed air may be passed through the small intestine of an infant in as few as ten minutes, especially if the subject is placed on his left side immediately after a meal, during which a considerable quantity of air is inevitably introduced into the stomach. The authors studied by roentgenograms a series of infants at alternate examinations: 1. They were encouraged to belch after the meal and then were put to bed lying on the right side. 2. They were placed immediately on the left side to favor the passage of swallowed gas through the pylorus. The roentgenograms of group 2 showed an appreciably larger quantity of gas in the small and large intestines. No relationship of meteorism to symptomatology was noted in this series.

McINTOSH, New York.

SERUM CALCIUM AND PHOSPHORUS IN RELATION TO DENTAL CARIES. I. JUNDELL and H. MAGNUSSON, Acta pædiat. **14**:13 (Nov.) 30) 1932.

The blood of twenty children with advanced caries was compared with that of twelve caries-free controls. The serum calcium and phosphorus were essentially the same in both groups.

McINTOSH, New York.

FURTHER EXPERIENCES WITH ISOLATION TENTS. I. JUNDELL, Acta pædiat. **14**: 172 (Nov. 30) 1932.

Portable cubicles for use in wards have been described previously by the author (*Acta pædiat.* **8**:375, 1929). With further technical improvements, set forth in this communication, he continued to find the cubicles satisfactory substitutes for more formal equipment. They effectually limited the spread of measles and pertussis, even when they were used by the routine nursing services and not manned by a specially trained and segregated personnel.

McINTOSH, New York.

ADALBERT VON CZERNY, SEPTUAGENARIAN. C. DE LANGE, Maandschr. v. kindergeneesk. **2**:309 (April) 1933.

The author gives an enthusiastic description of the life and work of Czerny and of their significance in pediatrics on the occasion of his seventieth birthday, March 25, 1933.

VAN CREVELD, Amsterdam, The Netherlands.

Society Transactions

NEW YORK ACADEMY OF MEDICINE, SECTION OF PEDIATRICS

Regular Meeting, Nov. 9, 1933

MARTHA WOLLSTEIN, M.D., *Presiding*

THE VALUE TO A PEDIATRIC CLINIC OF AN INTRAMURAL PSYCHIATRIC CLINIC. DR. EDWARDS A. PARK, Baltimore.

The Harriet Lane Home owes its psychiatric clinic to the interest of the Josiah Macy Jr. Foundation. It is under the charge of Dr. Leo Kanner, who is an associate professor in the department of psychiatry. The clinic is located in the Harriet Lane Home and, though belonging to psychiatry practically speaking, it has been as much an integral part of the Harriet Lane Home as the clinic for children with heart disease or that for tuberculous infants. The department of psychiatry has only outpatients.

All patients entering the psychiatric clinic to be seen by Dr. Kanner and his associates are referred from the pediatric clinic and have already been investigated completely by the pediatricians. When the investigation conducted by the psychiatric clinic is completed, a full report is written and attached to the unit history and through that medium is brought to the attention of the pediatrician who referred the case to the clinic. On receiving the report from the psychiatric clinic the pediatrician undertakes treatment with Dr. Kanner's knowledge, or Dr. Kanner undertakes the treatment with the pediatrician's knowledge, or they cooperate in the treatment. The pediatrician takes charge of the patient if he can and wishes to do so. The cases which the psychiatric clinic takes over are the more difficult ones, requiring special insight, guiding skill or much labor and time or special command of the community machinery for the care of problem children.

During the three years of its existence the psychiatric clinic has taken charge of 1,400 children. It is roughly estimated that among the new patients coming to the dispensary 1 out of 10 brings some problem needing adjustment in the psychiatric clinic.

Dr. Kanner has taken pains to make clear to the pediatric staff that no disturbance or difficulty in behavior or, indeed, variation from normal behavior can be too slight for his interest or aid. In consequence, children are sent to him because of lack of appetite, vomiting, rumination, disturbances of sleep such as night terrors and sleep walking, disturbances in speech, bad habits such as tics and nail biting, disturbances of a sexual nature, antisocial exhibitions, emotional upheavals, hysterical conditions and mental defects.

The value of the psychiatric clinic in the Harriet Lane Home lies chiefly in (1) the care of individual children, (2) the education of physicians and students and (3) the investigation of abnormal behavior in the human being. Probably no experiment in the Harriet Lane Home has had a more stimulating effect or greater usefulness or has exerted a greater educational influence than the intramural psychiatric clinic.

THE PSYCHOBIOLOGIC APPROACH TO THE DIFFICULTIES IN CHILDREN'S PERSONALITIES. DR. LEO KANNER, Baltimore.

The modern pediatrician has come to appreciate more and more the need of becoming familiar with the mechanisms and the handling of the common disorders of children's personalities. In the quest for adequate information one has been

bewildered and deterred by the different sets of nomenclatures, by the variety of psychologic systems with one-sided emphasis on one factor or another, and by the controversial battle between the adherents of the different schools. One has been baffled by a "confusion of theory and authority" (Brennemann) making it difficult to decide whether one should subscribe to the focal infection theory, psychoanalysis, Adlerianism, behaviorism or any other "school."

The psychobiologic approach assumes the natural common sense attitude. It works with the child as a whole, as a behaving human totality, whose functions as a whole are not less accessible to concrete and objective study than are the individual functions of his organs and system. It does not contain anything dogmatic and does not require ceremonious technics. It can be learned by any intelligent and conscientious physician who is willing to work not only with detached organs and their diseases, but also with persons and their troubles. The child is studied from the angles of his physical, intellectual, emotional, environmental and constitutional peculiarities. Psychiatric treatment is viewed as the total of the efforts made in behalf of the adjustment of a person with difficulties of personality; it may include work on physical repair, correction of faulty training in habits, doing away with harmful notions or attitudes of the child or of his family, better adaptation at school and many other measures, depending on the requirements of the patient. The therapeutic program considers not only the child, but also the environment, especially the parents and, whenever indicated, the teachers, recreational centers and other community agencies.

At the Harriet Lane Home, during the past three years, an intramural psychiatric department has become an integral part of the pediatric service and teaching, as the psychobiologic approach has proved to be acceptable and helpful to the pediatricians.

PERSONALITY DIFFICULTIES IN THE PRESCHOOL CHILD AND A METHOD OF DEALING WITH THEM IN A NURSERY GROUP. RUTH W. WASHBURN, New Haven, Conn.

Preschool children with difficulties in behavior or personality, brought for consultation to the Clinic of Child Development, Yale School of Medicine, are invited to join, on a prescription basis, a small group of normal, well adjusted children of similar chronological age, who are in daily attendance in the nursery. Methods of management and attitudes in situations arising in child care can be graphically demonstrated to parents whose children are members of this social group. Parents can watch their children through a one-way-vision screen, which is so arranged as to make the adults invisible to the children. With psychologic distance between parent and child thus assured, it is often possible to demonstrate dramatic changes in the child's behavior. After much observation of the child, the parents are sometimes able to solve their own problems of management without further discussion. The child's attendance in the nursery continues until improvement in behavior indicates that there is no further need for it. If the difficulty is based on emotional complications in the family, rather than mere ignorance of the laws of child psychology, the parents are referred to a psychiatrist.

Certain forms of behavior traceable to factors in personality come up for discussion in almost every interview with the parents of preschool children. For example, because of the inevitably close bond between parent and child, children are prone to behave more expressively with their parents than with other people. This expressive behavior is apt to be taken too seriously; its emotional significance to the child is overinterpreted. Again, children tend to continue to behave in a way that is useful to them. If a temper tantrum produces the desired result, it is worth while to continue to make use of so potent a weapon. A third point is that most preschool children go through a negative phase. "No," even in the mouth of a 2 year old, is a powerful word, which makes his opponent feel helpless. If the laws and prohibitions surrounding a child are unnecessarily multiplied, if frequent commands are given, this negativism is accentuated and a rebellious

child develops. Lastly, young children crave attention and would often rather have unpleasant attention, such as a spanking, than none.

One is constantly impressed by the marked effect which emotional factors may have on a child's development. N. J. was a sensitive child whose family situation was disturbed throughout his early life. At 2 years and 8 months of age he presented a picture of retarded mental development. He was not talking, and it was hard to elicit his attention. His behavior was egoistic and asocial in the extreme. A psychiatrist helped the parents to make a better adjustment. The child attended the nursery group for a period of months. At the age of 5 years his behavior reflected the improvement in the general situation. The developmental examination revealed superior intelligence. The child's vocabulary and use of words were unusually good. The asocial behavior was markedly modified. He proved to be an affectionate child, very dependent on rapport with the people to whom he was devoted. His interests have consistently been highly specialized.

L. D. was brought to the hospital at the age of 17 months in a state of serious malnutrition. No adequate physical cause for her condition was found. The child of parents whose personalities were of dramatically opposed types, her condition, in part at least, resulted from the disturbed conditions in the home. Early precocious development was reported, but the rate of mental development was slowed up when she was studied at the height of her illness at the age of 20 months. Her attitudes, particularly toward food, were much improved by the nurses' methods of management. The psychiatrist helped the parents to make a better adjustment to one another. They were able to care for the child more consistently and less emotionally. When seen at 32 months of age, the child's physical condition was markedly improved, the developmental rate was accelerated twenty points and she was relatively well adjusted with respect to personality. Like her mother, she was extremely sociable and reacted quickly to the moods of the people who cared for her.

HARRY BAKWIN, M.D., *Secretary*

Regular Meeting, Dec. 14, 1933

MARTHA WOLLSTEIN, M.D., *Presiding*

DR. ALFRED FABIAN HESS. DR. ROWLAND G. FREEMAN, SR.

In the death of Dr. Alfred Fabian Hess on the evening of Tuesday, December 5, the medical profession suffered a serious loss. He was only 58 years old and to most of those who knew him appeared to be a robust middle-aged man. His sudden death was thus a great shock.

The son of a publisher, Dr. Hess was graduated from Harvard University and then attended the College of Physicians and Surgeons of New York, graduating in 1901. He served as intern at Mount Sinai Hospital, and studied for several years in Prague, Vienna and Berlin.

He had an active imagination controlled by a judicial temperament which resulted in the accomplishment of a large amount of valuable research, the published reports of which number 223. Many of these publications carry several names, for Dr. Hess collaborated well with other men, and in so doing he provided for a continuance of his work by the younger men whom he trained.

While he will always be remembered as an important figure in the study of vitamins, his work had a much greater range than this. His early publications were on general clinical pediatrics, his earliest work showing considerable interest in the study of tuberculosis and its dissemination. He was instrumental in founding

the Tuberculosis Preventorium for Children and was always a leader in the administration of this valuable institution. His research covered the contagious diseases of childhood, the contents of the stomach, hemophilia and blood coagulation. Since 1920 his work has been centered particularly on rickets and scurvy and their control, and he has contributed books on these subjects.

All of his discoveries have received recognition by the medical profession both here and abroad. He was a member of the American Medical Association, New York Academy of Medicine, American Pediatric Society, American Philosophical Society, Society for Experimental Biology and Medicine, Association of American Physicians and numerous other societies. He was an honorary member of the German Pediatric Society and the Society of Pediatric Research and president of the Harvey Society. In 1927, he was awarded the John Scott Medal by the City of Philadelphia "for a method of producing a vitamin factor in food by ultraviolet light." In 1930 he received the honorary degree of Doctor of Science from the University of Michigan.

He was Clinical Professor of Pediatrics at the University and Bellevue Hospital Medical School, where I came in contact with him during the period when I held the chair of pediatrics there. I found him to be always a reliable teacher of sane pediatrics. At the Home for Hebrew Infants Dr. Hess did a great deal of valuable clinical work, and this institution owes much to him.

From his wide research in medical subjects in which he was interested Dr. Hess produced each year new discoveries. These were always presented with a judicial estimate, so that one could depend absolutely on his conclusions. In the death of Dr. Hess the medical profession has lost not only an excellent clinician and a reliable teacher, but a valuable investigator as well.

SERUM PHOSPHATASE AS A CRITERION OF THE SEVERITY AND OF THE RATE OF HEALING OF RICKETS. DR. AARON BODANSKY and DR. HENRY L. JAFFE.

This article will appear in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

SERUM PROTEIN DEFICIENCY WITHOUT REVERSAL OF THE ALBUMIN: GLOBULIN RATIO. DR. A. A. WEECH.

The commonly encountered disorders associated with a diminished amount of protein in the serum are chronic Bright's disease and malnutrition. Edema may be a symptom of both conditions when the deficit of serum protein is sufficiently great. In both disorders the albumin fraction of the serum generally suffers greater depletion than the globulin fraction; indeed, the globulin may remain at a normal level or rise. The result is that the normal ratio of the albumin to the globulin is reversed and values less than unity are the rule rather than the exception. Within the last year and a half two unusual cases of edema have been reported in which hypoproteinemia was associated with a normal ratio of albumin to globulin. One of these occurred in a girl 2½ years of age (*Tr. Am. Pediat. Soc.* **44**:30, 1932) and the other in a man (*J. A. M. A.* **101**:198 [July 15] 1933). In both, the cause of the deficit of plasma protein was obscure and the authors independently suggested the possibility of a defect in the synthesis of the proteins within the body.

This circumstance has instigated a review of the analyses of serum in a group of fifteen children with nephrotic edema and in a group of twenty children with nutritional edema. The data collected are presented in the table. In the nutritional group the albumin: globulin quotient was less than unity in fourteen cases and greater than unity in six cases, that is, the normal ratio was reversed in 70 per cent and not reversed in 30 per cent. In the nephrotic group only one patient showed a ratio with a value greater than unity, that is, there was reversal in 93 per cent and no reversal in 7 per cent. In the single instance of nonreversal the value of

the ratio is at the lower limit of normal. The relatively lower values of the ratios in nephrotic edema undoubtedly result from the large selective loss of albumin in the urine. It is clear, however, that absence of reversal of the ratio of albumin to globulin is not a rare finding in nutritional edema.

How, then, is one to recognize cases of edema which seem to depend on a defect in the ability of the body to synthesize serum protein? It is apparent that the presence of albumin and globulin in normal proportions in the serum can do no more than point the way to further study. Observation of the effect of feeding an adequate amount of protein will provide evidence of considerable negative value, for in most instances nutritional edema disappears and the serum proteins return to the level of health. Occasionally, however, the basis of nutrition edema appears to be defective absorption rather than deficient ingestion of protein and in such cases the condition is not relieved by an adequate diet. In the reported cases of defect in the synthesis of serum protein diagnosis has rested on studies

Determinations of Serum Protein in Children with Edema

Nutritional Edema				Nephrotic Edema			
Albumin, per Cent	Globulin, per Cent	Total Protein, per Cent	Albumin / Globulin	Albumin, per Cent	Globulin, per Cent	Total Protein, per Cent	Albumin / Globulin
1.63	3.46	5.09	0.47	0.69	2.86	3.55	0.24
1.27	2.70	3.97	0.47	0.93	2.91	3.84	0.32
1.62	3.31	4.93	0.49	0.83	2.36	3.19	0.36
1.68	2.89	4.57	0.58	1.03	2.83	3.86	0.36
1.03	1.73	2.76	0.60	1.11	3.13	4.24	0.36
1.88	3.13	5.01	0.60	0.54	1.46	2.00	0.37
1.28	2.10	3.38	0.61	0.92	2.38	3.30	0.39
1.25	1.81	3.06	0.69	1.41	3.39	4.80	0.42
1.10	1.51	2.61	0.73	1.04	2.31	3.35	0.45
1.77	2.32	4.09	0.77	1.84	3.62	5.46	0.46
1.18	1.52	2.70	0.78	1.14	2.41	3.55	0.47
2.14	2.54	4.68	0.84	1.78	2.10	3.88	0.85
1.07	1.25	2.32	0.86	1.23	1.29	2.52	0.95
2.21	2.29	4.50	0.97	1.43	1.46	2.89	0.98
1.64	1.61	3.25	1.02	1.75	1.41	3.16	1.24
1.69	1.33	3.02	1.27				
2.06	1.62	3.68	1.27				
2.54	1.81	4.35	1.40				
2.97	1.53	4.50	1.96				
2.86	1.44	4.30	1.98				
1.66*	1.99	3.65	0.83	1.11	2.28	3.39	0.49

* The mean values are geometric means.

of nitrogen balance, by means of which it was demonstrated that the serum proteins remained low in spite of an intake of food sufficient to establish a positive nitrogen balance.

STUDIES IN PROPHYLAXIS OF MEASLES. DR. BÉLA SCHICK and DR. SAMUEL KARELITZ.

An attempt was made to increase the existing immunity of adults to measles by reexposing them to active measles from eight to thirteen times within a period of two weeks. Blood serum obtained prior to the reexposure and again eight and twenty-one days after the last reexposure was tested prophylactically. The results suggested a slight increase of the immunity after reinfection, but this was not striking enough to be considered definite.

Comparison of patients treated in institutions with those treated at home showed a much greater percentage of complete protection in institutions than in homes. Further comparison of persons treated in homes in which isolation of the sick sibling was possible with those treated in homes in which the contact between the sick and susceptible siblings was uninterrupted showed a much greater percentage of complete protection in the former group. When the dosage of serum for the

patients treated at home was increased, there was a corresponding increase in the percentage of complete protections and consequently a smaller variation from the results obtained in children in institutions.

As in tuberculosis, the doses of infection obtained in exposures to measles help to determine the percentage of infections, and this suggests the necessity of using larger doses of serum when complete protection is desired and the exposure is severe. The globulin fraction of some of the serum was tested. The results indicated that this fraction was at least as effective as, and probably slightly more effective than, the serum from which it was extracted.

DISCUSSION

DR. BÉLA SCHICK: It is reasonable to assume that repeated exposure to diphtheria or measles may help in keeping up the immunity against these diseases for the entire life. In a city the size of New York every one has abundant opportunity to be exposed to measles. Our studies show that it is important that the child should not be exposed to superinfection during the period of incubation. Superinfection after antibodies are established may be less dangerous. This fact is of importance not only for the control of measles but also for that of tuberculosis and pertussis.

SERUM BASES OF INFANTS WITH ACUTE INTESTINAL INTOXICATION. DR. AUBREY B. MACLEAN and RUTH C. SULLIVAN, PH.D.

Deficit in the serum bases and loss of water are usually conceded as characteristic of acute intestinal intoxication. The therapy, therefore, has taken the form of administration of salt and water. It was believed that a knowledge of the variation of the individual bases—sodium, potassium, calcium and magnesium—in the blood serum might be of aid in providing a more intelligent approach in treatment. No record has been found in the literature of the simultaneous analysis of the serum of infants with acute toxic diarrhea for the individual bases—sodium, potassium, calcium and magnesium.

We present data on the serum of fifty-seven infants. The total serum base, or the sum of the sodium, potassium, calcium and magnesium, is given for each of forty-five infants with acute intestinal intoxication. Thirteen (29 per cent) showed a total base within the normal range (from 152 to 158 milliequivalents), eleven (24 per cent) showed a total base below normal and twenty-one (47 per cent) showed an increase in the total base. Determinations of the serum sodium are given for forty-seven infants. Nine determinations (19 per cent) were in the normal range (from 139 to 144 milliequivalents); 38 per cent were below and 43 per cent were above the normal range. Serums from fifty-five infants with intestinal intoxication were analyzed for potassium. In sixteen serums (30 per cent) the content of potassium was in the normal range (from 5 to 7 milliequivalents); in nineteen (33 per cent) it was below normal and in twenty-one (37 per cent) it was above normal. Calcium was determined in the serums of forty-two infants. With the exception of one value slightly below and three slightly above, all values were in the normal range. Magnesium was determined in the serums of forty-two infants. According to our figures for normal controls, the range of which was from 1 to 2.08 milliequivalents, twelve (29 per cent) of the serums showed values for magnesium greater than normal. According to the figures given in the literature, all the determinations fall within the normal range.

The idea has been advanced that the concentration of total base in the blood plasma is one of the most carefully guarded constants of the body. Any attempt at change in the total electrolyte content is stubbornly resisted. In fact, it is even difficult to produce marked changes experimentally. This does not seem to be true in infants with acute intestinal intoxication. Marked variations from the normal are seen in both directions.

It has also been pointed out that retention of base means retention of water. This does not seem to be true in infants with intestinal intoxication. Marked

elevations of the serum base beyond normal limits were effected without improvement in the dehydrated condition of the infants. When a neutral salt is introduced into the organism in a concentration greater than is normal in the plasma the ion in excess, either acid or basic, is excreted by the kidneys and the normal composition of the plasma is restored (Peters and van Slyke). This is not true in infants with intestinal intoxication. When sodium chloride was given to such infants, in whose serum there existed a deficit in base with a relative increase in chlorides, it was expected that the sodium would be retained and that the chloride would be excreted by the kidneys as ammonium chloride. On the contrary, an increase of both sodium and chloride was found in the plasma after a subcutaneous injection of sodium chloride.

Just as the study of diabetes has been of inestimable aid in advancing knowledge of the physiology of the pancreas, so the study of intestinal intoxication is contributing much to the understanding of the salt and water metabolism in the body.

DISCUSSION

DR. BENJAMIN KRAMER: Several months ago, Gamble (*J. Pediat.* **3**:84 [July] 1933) reported some studies on the relation of mineral excretion to water excretion in infants with acute diarrhea. He attempted to determine the source of the water which is lost in this condition, i. e., whether the source is extracellular or intracellular. Since the sodium in extracellular water is very high in proportion to the potassium, a similar ratio should be found in the excreted water if the latter is derived solely from extracellular water. If, however, the excreted water is derived from intracellular water, the potassium should be definitely high in proportion to the sodium. Analysis revealed an excess of potassium in the excreta.

In searching for an explanation of this excess of potassium in the excreta, Gamble suggested that the concentration of potassium in the serum of infants with acute diarrhea with intoxication may be much higher than in normal infants. This would explain the low sodium in proportion to the potassium in the excreta. In a few cases which my associates and I have studied, the potassium content has been either normal or moderately reduced. The studies of MacLean and Ruth Sullivan indicate clearly that at times the potassium in the serum and presumably that in the fluid of the tissues may be very high. It is reasonable to suppose that Gamble was studying this type of case.

STUDIES IN AMYLOIDOSIS. DR. HAROLD G. GRAYZEL, DR. MENDEL JACOBI, DR. HYMAN WARSHALL, DR. MAXWELL BOGIN, DR. HERMAN BOLKER and DR. BENJAMIN KRAMER.

The disappearance of a marked enlargement of the liver and spleen (these reached into the pelvis), the disappearance of ascites, the diminution in albuminuria and the improvement in the anemia in proved cases of amyloidosis complicating tuberculosis of the bone after liver therapy led to an experimental study of the subject. Amyloidosis was easily produced in white mice by subcutaneous injection of sodium caseinate solution (nutrose). The first amyloid demonstrable pathologically appeared as crystalline masses within the macrophages, both wandering and fixed, in the spleen and liver. These masses increased in size, lost their crystalline character in becoming homogeneous, brought about cellular necrobiosis and fused with other similar masses, originally intracellular, as well as with concomitantly developing extracellular amyloid. In the late stages the amyloid masses caused marked compression atrophy of the normal parenchymal tissues.

Amyloid regularly was demonstrable in white mice fed a stock diet of bread and oats (3:1) after thirty daily injections of sodium caseinate solution. Administration of ground beef, cholesterol or diets with acid or alkaline residue produced no retarding or accelerating effect. Two liver extracts added to the diet exerted a slight retarding effect (the first signs after forty-two injections). Powdered whole liver (10 per cent of the diet) added to the stock diet retarded the production of amyloid until after sixty-six injections of sodium caseinate solution had

been administered. Thereafter the amount of amyloid lagged far behind that produced in animals not treated with liver. A similar but less marked inhibiting influence on the production of amyloid was evidenced by a combination of vitamins A and B added to the stock diet (each was 5 per cent of the total diet). No additional effect was noted on the addition of other vitamins.

Spontaneous resorption of experimentally produced amyloid does not occur. The addition of powdered whole liver to the stock diet of mice in which amyloid has been produced causes the resorption and total disappearance of the latter in cases of slight, moderate and moderately advanced amyloidosis. It produces a diminution of the amyloid, but not its total disappearance, in cases of severe, far advanced amyloidosis. The disease is considered a metabolic rather than a degenerative disease. It is reversible.

Ten cases of amyloidosis have been studied in detail. Four patients who had this disorder in severe form have been without clinical evidence of amyloid for periods of from two to four years, after treatment with powdered whole liver for periods varying from eight to fourteen months. Three with severe amyloidosis are still under observation from six to twelve months after the institution of the treatment; all show signs of marked regression of the disease, are free from symptoms, but still present palpable (but much smaller) spleens and livers and slight albuminuria. Three patients with the disease in very severe form died of intercurrent infections within eight, nine and eleven months after the institution of treatment. Treatment in their cases was spasmodic and unsatisfactory because the patients refused to take or could not retain liver preparations. It is interesting to note that the regressive changes in the organs occurred despite the continued presence of inciting infections. Further, the first signs of regression appeared only after protracted therapy (for at least from eight to ten months); the regressions were gradual at first, but were rapidly accelerated in the cases with favorable prognosis, finally continuing, in the absence of liver therapy, to the total disappearance of signs. This phenomenon suggests a cumulative stimulative effect on the organism, probably on the reticulo-endothelial system, which is predominantly involved in the experimental disease.

DISCUSSION

DR. LESLIE O. ASHTON: If a diagnosis of enlargement of the liver were made, what would the enlargement be due to in those cases in which the liver shrank? What was the etiologic factor in the enlargement of the liver in the children reported?

DR. MENDEL JACOBI: All these were proved cases of amyloidosis, as demonstrated by the clinical picture and urinary findings and confirmed by positive results from congo red tests (Bennhold). Again, no other cause for hepatic enlargement was present. In those cases in which the liver shrank, we think that the shrinkage was due to resorption of the amyloid, a fact we demonstrated experimentally. In the one unsuccessfully treated patient who died and whose liver remained large after shrinking somewhat, an intense cirrhotic reaction may have been present; the liver felt nodular and harder than the others, and the ascites, which at first diminished, recurred and remained unaltered by further therapy.

DR. X: A puncture of the spleen was not done. There is nothing to indicate amyloidosis but enlargement of the liver and spleen. This is insufficient to indicate amyloid disease. Enlargement of the liver by fat is seen frequently in tuberculosis, which has nothing to do with amyloidosis.

DR. MENDEL JACOBI: No punctures of the liver or spleen were made. The clinical picture, however, of generalized amyloidosis is striking, so that once seen it is not easily forgotten, and in all our cases the presence of the clinical picture was confirmed by the congo red test. While it is true that the liver may be enlarged without amyloid involvement in cases of chronic tuberculosis such enlargement is usually very slight, the liver being somewhat palpable beneath the costal margin. Further, its edge is not usually hard or rounded. In our cases the organ extended into the pelvis and was very hard and rounded.

The diagnosis of fatty infiltration is, at best, doubtful clinically in reference to any organ and pathologically is being discarded. In discussing disease of the liver the term has no place, and fat phanerosis does not produce enlargement of the liver comparable to that caused by amyloid disease. Besides, fat phanerosis is not very marked in cases of chronic tuberculosis of the bone. Finally, I have never heard of splenic enlargement by fatty inclusions as a complication of tuberculosis; nor have I, in several thousand autopsies, seen splenomegaly in this disease except in association with amyloidotic complications.

CHICAGO PEDIATRIC SOCIETY

Regular Meeting, Nov. 21, 1933

CHARLES SCHOTT, M.D., *President, in the Chair*

FOOD FACTORS IN ALLERGY OF CHILDHOOD. DR. GEORGE PINESS, Los Angeles (by invitation).

DISCUSSION

DR. I. HARRISON TUMPEER: I am fervently in agreement with everything that Dr. Piness has so concisely stated. To the clinical observations that foods may produce respiratory symptoms and that inhalants and pollen may cause intestinal symptoms I nod assent. To the fact that many patients with hay fever do not get the maximum benefit from treatment because the cumulative dosage of pollen plus food and inhalants is ignored I say, "Amen." To the necessity for complete testing I shout, "Hallelujah!" In too many places the question, "Who tests for allergy?" provokes the betraying response, "Every one does a little testing." But because "every one" fails to live up to the dictates which Dr. Piness has emphasized, allergy often falls into disrepute. The enthusiasts in the study of allergy are not physicians in the authoritative position of Dr. Piness. He is an optimist but not an enthusiast in the sense of crusading for untenable claims. The enthusiast is the amateur who does a few tests, closes his eyes to the rest, concludes that the cutaneous reactions form the whole story and proceeds blindly on the basis of his few tests alone. If his patient fails to respond, he believes that there is "nothing to" allergy. I am reminded of an eastern amateur in the treatment of eczema, who has decided that reactions to egg are nonspecific because so many babies with eczema who have not eaten eggs show positive reactions. Many of us have had the experience of meeting physicians who "do a little testing" in private and decry the implications of allergy in public. If no one is around they ask eagerly for the latest information on the technic of testing and treatment.

That brings me to the questions: "Who is the qualified allergist?" and "What are the makings of an allergist?" I shall take my friend, Dr. Piness, as an example. Allergy was developed in its modern aspects by a group of eastern clinicians. After Dr. Piness worked with one of these pioneers, Dr. Schloss, he went west and decided to be the complete allergist that he is now known to be. Working toward this end he "botanized" to learn the flora of the west. Then he acquired a field botanist for this work and employed another botanist with a master's degree in his office. Soon the importance of food factors necessitated having a dietitian on his staff. In his paper he mentioned the kinds of testing material. That statement covered a lot of ground. For the preparation of his extracts he employs a doctor of philosophy in physiologic chemistry, a man of scientific and practical parts. Gordon Alles was originally engaged in freeing insulin from protein material. His present job requires the extraction of pure proteins for Dr. Piness' allergic practice. Alles is of university caliber, like the rest of the staff, and has been loaned to one of the university commissions to purify liver extracts. From the scientific debates in the laboratory between Dr. Piness, his associates, Dr. Miller,

and Alles, one might conclude that this group was engaged in destroying painstakingly gained ideas about allergy instead of critically assaying the facts.

What authority has Dr. Piness to give a talk covering a clinical subject in children's allergy? Let me take you on his daily rounds. We begin with one of the two medical divisions of the Cedars of Lebanon Hospital. Dr. Piness is chief of this service and is a member of the executive committee presiding over the outpatient department. This compels contact with clinical medicine. Another day we arrive at the Hospital of the Good Samaritan at 8 a. m., where some 200 adults are attending the clinic for allergy. We have yet to visit the most significant of all. We arrive at the Children's Hospital about 1:30 p. m. twice a week. Try to imagine a clinic for children with allergy caring for from 80 to 125 children within ninety minutes, at most. Everything is tranquil. There is no confusion. You are mistaken if you are conjuring up in your minds the picture of a long line of bare arms and the staccato action of many needles. Every child referred to the clinic has been given a complete physical examination. Every new child has had his case reviewed by the resident physician the night before. All necessary consultations have been effected, including those with members of the laboratory staff, the roentgenologist, the nose and throat specialist and the dermatologist. I counted 14 persons working smoothly in the clinical hall. This group included Dr. Piness and his associate, technicians, nurses, orderlies and volunteers. Symbolic of tonight's subject, "Food Factors in the Allergy of Childhood," and of the importance accorded food factors in a clinic for children's allergy, the last contact the child makes during the session at the clinic is with the dietitian. This is a free clinic where every patient receives the same kinds of tests and treatment materials and the same number of tests as the private patient in Dr. Piness' office. The set-up of his office and that of the clinic are ideal.

To sum up, tonight the food factors in the allergy of childhood have been authoritatively elucidated.

DR. HARRY L. HUBER: Those who have heard Dr. Piness read a paper at any meeting marvel at the territory that he covers. I agree with what he has said, and the completeness with which he has covered the subject leaves little to be added. I should have liked him to say more on the subject of change of climate in the treatment of allergic patients. A change of climate is commonly recommended in these cases, but only a few patients are benefited by such a change. If the children sent to other climates had been completely studied in the manner described by Dr. Piness, most of them could have remained at home. The testing must be made with all foods and substances with which the patient can possibly come in contact. As Dr. Piness has emphasized, most patients are not aware of the great variety of foods that are commonly used.

DR. MILTON M. PORTIS: I wish to take this opportunity of expressing my appreciation to the guest speaker for coming so long a distance to bring his splendid message. I have been much interested in this subject, especially so because one of the members of my family has been suffering from allergic asthma.

There are many complex phases of this problem. Undoubtedly, a disturbance of the endocrine system may be an important factor. Environment and psychic disturbances are also to be considered. Inhalants deserve important consideration. Unquestionably, some persons are sensitive to particular foods, for when these foods are withdrawn the allergic disturbance is lessened or may disappear.

As Dr. Tumpeer has told you, Dr. Piness has developed an exceptionally able group, who work with him. These include a well qualified botanist, who is constantly studying the flora in his neighborhood, and an exceptionally competent chemist, who prepares the testing material and everything that is used in treatment. He has, besides, a skilful dietitian, who has a critical attitude, and who, at the same time, plans for enough suitable foods to keep the patient well nourished. Unfortunately, only few allergists in this country have such coordinated help, but to do thorough and scientific work such a group is almost necessary.

Some day, some one is going to be wise enough to answer the question, "Why allergy?" In my early days, volumes were written about the treatment of diphtheria, but as soon as antitoxin was introduced the whole subject was condensed into a few lines. It was merely necessary to state how much antitoxin to give and when to repeat the injection. I believe that some day some good physician who understands biology and chemistry will come along and explain why allergy exists, and then all the cumbersome technic and troublesome problems in connection with this condition will disappear. Until that time, God bless the pioneers, among whom the guest speaker deserves a prominent rank.

DR. MAX S. WIEN: To me, one of the outstanding points in Dr. Piness' excellent presentation is that he uses skin tests to confirm a diagnosis of allergy founded on a previous complete examination of the patient. He stressed diagnosis rather than testing, and I believe that this speaks for a much saner approach to the problem of the allergic patient.

On seeing children with eczema, I have been impressed with three types of cases. There is the infantile type, in which the eruption occurs on the face and a careful history reveals that it has come on following some change in the usual substances which come in contact with the infant's skin. If, after a careful history followed by removal of the irritant and the use of bland applications, the symptoms disappear, one is not much concerned with the allergic background.

The second group consists of cases in which the history reveals that the symptoms came on following contact with silk, wool, orris root, etc., or following such contact in combination with sensitivity to some article of diet, such as egg, a particular fruit or vegetable, wheat or milk; when these irritants are removed the condition clears up.

In the third group there is an associated involvement of the popliteal and anterior cubital spaces and of the sides of the neck, and the eruption changes in some instances from an acute erythematous oozing type to a crusted, scaling or licheniform variety. There may be a history of asthma in the family or in the patient. In these cases one is dealing with a serious allergic state, and it is necessary to confirm the diagnosis by a complete allergic investigation, such as that outlined by Dr. Piness.

DR. E. P. ZEISLER: I have had little experience with cutaneous allergy in childhood. I have followed Dr. Piness' work for many years, however, and have been much impressed by it. The attention of dermatologists has been directed to the importance of allergy particularly during the past few years. Bloch and Jadassohn in Europe and Sulzberger in this country have brought out several new points.

I should like to ask Dr. Piness what his experience has been with the propeptone therapy introduced by Urbach of Vienna. Urbach classified as alimentary idiosyncratic eczema those eczemas of childhood in which the cutaneous reactions are negative, and he believes that the offending allergen can be detected by oral administration of the specific propeptone. He differentiates eczema of this type from neurodermatitis, in the diagnosis and treatment of which skin tests are often of value. Few have adopted his suggestion in this country. Dr. Piness may be able to tell us more about this.

I have been disappointed in my own results in skin tests in allergic cutaneous disorders in childhood, but I am sure that this is because I am a novice and have not done the work as Dr. Piness insists it must be done. I think that, to do the work thoroughly, one must have the cooperation of an experienced allergist in addition to what the dermatologist has to offer.

DR. SAMUEL M. FEINBERG: There are one or two points that Dr. Piness did not mention which I should like to bring up, because I know that they are disputed. I often hear of children who supposedly have asthmatic bronchitis. That is a common diagnosis, but I am sure that Dr. Piness and other allergists will agree with me that the condition is rare in children. Just as an overdose of protein may precipitate an attack of asthma, so may acute respiratory infections precipitate allergic asthma. Also, what is frequently called bronchitis is in reality an allergic cough.

Another disputed point that I frequently encounter is: how early should these children be tested? Some say that one should wait until they are older. I see no reason why they should not be tested as soon as they show allergic symptoms. It is true that a year later they may be sensitive to different foods, but that is no excuse for not finding out what the fundamental causes are at the first occasion.

Another interesting feature that I have seen a number of times is a definite effect from the odors of foods cooking in the home. I have seen cases in which rhinitis or asthma was not entirely relieved until the offending foods were removed from the home.

Dr. Piness spoke of the difficulty of excluding foods from the dietary of children. The child may be breast-fed and show symptoms due to the mother's food, and it may be necessary to exclude certain foods from the mother's diet in order to relieve the child.

DR. LOUIS S. ROBINS: I think that many allergists in Chicago will do a much better business, beginning tomorrow, because half of them will stop testing their own patients. Dr. Feinberg mentioned that as soon as one sees a child with allergic manifestations one should test it. Is it advisable to test a child 3 months old who has eczema, and to how many test substances should one subject it? Also, should one do intracutaneous tests on a baby if the scratch tests are negative? In my limited experience I have seen positive results in only from 10 to 20 per cent of tests in children under 6 months of age. I have used only scratch tests on young infants. Would that account for the difference in the percentage of positive results, Dr. Piness having reported 70 per cent positive reactions? There are cases in which, if the child is put on goat's milk or modified cow's milk, the condition improves. In other cases eczema will clear up spontaneously when the child is 5 or 6 months old. Is there any justification, then, for carrying out all these tests on children before they are 6 months of age? Another question that I should like to ask is why asthma or even eczema clears up in some children when they are hospitalized. The accepted explanation is that this is due to removing the patient from the environment in which something is contributing to his condition. I have seen patients who have been shifted from one place to another, to Arizona and to California, and whose homes have been cleared of every possible irritant, with no improvement, who if placed in a hospital lose their eczematous condition. When they are taken home the symptoms reappear. Does the nervous condition of the child, of the mother and father and of the entire family aggravate the asthma, or is the condition due entirely to inhalants and food?

Book Reviews

Diet in Sinus Infections and Colds. By Egon V. Ullmann. Pp. 166. New York: The Macmillan Company, 1933.

This book, dedicated to the author's father, Dr. Karl Ullmann, is a worthy attempt to adapt the principles of diets used in other fields to the successful treatment of the common cold and of sinus infections. The diet, in the author's clear wording, "is directed to counteract acidosis, to increase the effect of calcium in the system and to prevent an eventual lack of vitamines."

There are twenty-one chapters, with an excellent bibliography. The first four chapters deal briefly and clearly with regional anatomy, general advice, food and civilization and the influence of various foods on human metabolism. Chapters 5 to 12, excluding chapter 6, treat of the influence of various proteins, carbohydrates, fats and condiments on acidity or alkalinity. Here one is advised of the high, unbalanced acidity of one's general daily diet as forced on one by present-day marketing. Particularly good are chapters 6 and 12. The former treats of the importance of an alkaline diet; in this chapter Berg points out the advantages of sufficient alkaline nutrition as against a disease-promoting acid-rich intake. The latter chapter is noteworthy in that it advocates a salt-poor diet rich in natural calcium. Here one is reminded that 1 Gm. of sodium chloride holds 70 Gm. of water and replaces and slows the assimilation of calcium, which plays such an important part in inflammatory processes. The restriction of salt counteracts inflammations, suppurations and exudations by the liberation of bound water. The enforcement of the action of calcium against inflammation by natural ways, Ullmann states, is one of the main principles of this diet. Vitamins are abundantly used in the juices of raw fruits and vegetables. Citrus juices precipitate calcium and therefore must not be taken until the latter is assimilated. Economy in the intake of water is shown to be highly advantageous.

The principles of this diet are summarized as follows: (*a*) Use only fresh food, (*b*) restrict salt, (*c*) prefer alkaline foods, (*d*) reduce animal proteins and (*e*) cut down carbohydrates and prefer unrefined to refined carbohydrates.

A list of forbidden foods, foods which can be taken in limited amounts and favored foods are given. Methods of preparing food are advocated. Frequent feedings are advised. Fruit and vegetable juice in various mixtures and preparations supply fluid and the essential food substances. Urinalysis is urged to check the reaction of the metabolism to the diet. The adoption of a restricted or partially restricted diet depends on the number of colds or sinus infections the patient has. The more common causes for failure in treatment are given and clearly dealt with, while the appendix contains many recipes and menus.

Handbuch der Roentgen-diagnostik und Therapie im Kindesalter. By Prof. Dr. St. Engle and Dr. L. Schall. Price, 75 marks, bound; 72 marks, unbound. Pp. 701, with 637 illustrations. Leipzig: Georg Thieme, 1933.

In this book on roentgen diagnosis and therapy in infants and children the authors have chosen collaborators to write some of the chapters on special subjects. The book is divided into two parts: roentgen diagnosis and roentgen therapy.

The introduction to the part on roentgen diagnosis consists of a complete discussion of the physics of roentgen rays and the methods used in their technical application to children. The chapters dealing with the diseases of the bony skeleton and the thorax are preceded by excellent discussions on the bony skeleton, including the age of appearance of the ossification centers, the growth of normal bones and

the development of the chest, with a complete discussion of the anatomy of the lymphatics. The following diseases of the skeletal system are thoroughly explained and beautifully illustrated: chondrodystrophia foetalis, cartilaginous exostoses and enchondromas, osteogenesis imperfecta, cretinism, mongolism, osteoporosis, rickets, scurvy, osteochondritis deformans juvenilis (including Calvé-Legg-Perthes' disease, Still's disease, Köhler's bone disease and Osgood-Schlatter's disease), diffuse osteosclerosis, osteitis fibrosa cystica, congenital syphilis, tuberculosis, osteomyelitis and others. The chapters on the diagnosis of the diseases of the thorax are equally complete, including a discussion of congenital anomalies and of the specific and nonspecific diseases. Tuberculous infections of the lymph glands and lungs and congenital cardiac defects are especially well illustrated. Other subjects equally well outlined are the gastro-intestinal tract, the urogenital system and the nervous system (including a chapter on the technic of encephalography).

The introduction to the second part of this book includes a discussion of the physics of the rays used in therapy, their action on the organs of the body, the dosage and the methods of application. In the chapters on roentgen therapy, the treatment of tuberculosis and diseases of the hematopoietic and lymphatic systems (including the lymphatic tissue of the nasal pharynx and the tonsils), the action of roentgen rays on the glands of internal secretion and the treatment of eczema, asthma, tumors and similar diseases are discussed.

This is a valuable reference book for physicians interested in roentgen diagnosis and roentgen treatment of diseases of infancy and childhood, because of its completeness and its excellent illustrations.

Traité de médecine des enfants. Volume I. By P. Nobécourt, L. Babonneix, J. Cathala and J. Hutinel. Price, 170 francs. Pp. 884, with illustrations. Paris: Masson & Cie, 1933.

This beautifully bound and published book is the first volume of a system of pediatrics in the French language. The introduction contains a short history of French pediatrics, with portraits and illustrations from art of pediatric subjects, a most interesting addition to any book.

The body of the work starts with a chapter "Normal Growth," which is detailed and up to date. The striking feature of this volume is the next chapter and the ones following it, which have to do with disturbances of growth. This is taken up in a way quite different from that in which the subject is considered in this country and contains much material on disturbances associated with changes in the ductless glands. These chapters are most interesting, well prepared and well illustrated.

The next chapters take up the question of malnutrition and overnutrition and are written in the same comprehensive fashion. Next comes a chapter on diabetes, then one on diabetes insipidus and one on arthritism, a condition which American physicians are loath to accept as a separate entity or even as a diathesis. Then come an excellent chapter on chronic articular rheumatism and a short one on gout. Chapters on cellulitis, progressive lipodystrophy and prematurity follow. The chapter on prematurity is quite complete. Then come chapters on sclerema and edema of the new-born and on angioneurotic edema.

The next large chapter is that on congenital diseases, which is followed by chapters on conditions due to a deficient diet. These are taken up in a serial manner and include not only avitaminoses but also deficiencies of the organic and inorganic constituents of food. These are followed by a large and excellent chapter on rickets, and this in turn is followed by several chapters on the ductless glands.

The volume impresses one as a peculiarly practical treatise on the subjects named, in which it is easy to find items in which one may be particularly interested. The book is well illustrated where illustrations are necessary. There are no colored illustrations. It will unquestionably be a valuable addition to any pediatric library.

Traité de médecine des enfants. Volume II. By P. Nobécourt, L. Babonneix, J. Cathala and J. Hutinel. Price, 150 francs; bound, 170 francs. Pp. 958, with illustrations. Paris: Masson & Cie, 1933.

This is volume II of the new French system of pediatrics. It is confined largely to the treatment of contagious and infectious diseases. There are short chapters on anemia and diseases of the lymphatic glands. The work is illustrated well but not profusely. The chapters on diphtheria are especially extensive and good, but the outstanding feature of the volume is the discussion of congenital syphilis by Péhu. This comprises approximately 150 pages, and the subject is taken up from every angle. This chapter is a monograph in itself by a pediatrician who is perhaps the best known authority in the world on this subject. The chapters on anemias are rather disappointing. The rest of the book, however, in which many of the rarer oriental infectious diseases are considered, is valuable. There are three colored illustrations in this volume which are especially good.

Los conceptos sobre la constitución individual y la fisiopatología del lactante. By Anibal Ariztia. Pp. 189, with 48 figures and photographs. Santiago: Nascimento, 1933.

The author of this book is professor of pediatrics of the University of Chile. It is his conception that individual physiologic conditions existing in the germ plasm may affect the offspring. The first chapter deals with such known and acknowledged factors as achondroplasia, cretinism and congenital deformities. In the second chapter, the author, using the classification of Sigaud, shows how these types are inherited, with the different phases of their mixture. In the third chapter he deals with the development of dystrophies, debilities and prematurity, and in the final fourth chapter with functional alterations in the organism that are derived from constitutional anomalies, such as exudative diathesis, rickets, avitaminosis and similar deficiencies. This work is well written and profoundly interesting. The photographs are well reproduced, and the printing is excellent.

The Therapeutics of Malaria. Bureau for Increasing the Use of Quinine. Amsterdam, the Netherlands, 1933. Pp. 19.

Some of the principal statements of the Malaria Commission of the League of Nations, as published in their third general report (*Quarterly Bulletin of the Health Organization of the League of Nations*, vol. 2, no. 2, June, 1933), are discussed in this booklet.

In only two cases should preference be given to drugs other than quinine, viz., to atabrine, in treating primary attacks of malignant tertian fever, and to plasmochin, in destroying the gametocytes (crescents) of the same type of malaria. In other cases in which drugs are effective quinine has stood the test with the new synthetic drugs. The abundance, availability and low price of quinine make it of especial value for use on a large scale in malarious countries.

Crisoterapía de la tuberculosis. By Luis Sayé. Paper. Pp. 182, with 151 photographs. Barcelona: P. Salvat, 1933.

This book deals with the treatment of tuberculosis with chemotherapy. The various gold preparations are discussed in detail, with particular attention to "Sanocrysin." This substance the author believes to be most efficacious. The literature concerning the results of this form of treatment has been collected and studied. All the case reports are beautifully illustrated with roentgenograms. In the study of his own results the author has taken a very fair view. Briefly, he believes that treatment with gold compounds is of value in infantile tuberculosis and in initial pulmonary tuberculosis of the adult, particularly when combined with col-

lapse therapy. This book is excellently written; the illustrations are remarkably clear, and the printing is perfect. It seems to the reviewer that an English translation would be of value.

La sténose hypertrophique du pylore chez le nourrisson. By J. Poucel. Price, 20 francs. Pp. 102, with illustrations. Paris: Masson & Cie, 1934.

This short monograph of 102 pages is complete from a clinical standpoint. It is evident that Poucel knows practically nothing about the literature on the subject except what has been published in France. The references and text show a decided narrowness of outlook. On the other hand, the material is well handled, and the subject is fully discussed.

While he does not cite American literature, Poucel evidently knows enough about it to criticize it for not giving Fredet credit for the operation which in this country is commonly known by the name of Ramstedt.

To one familiar with the subject, the book affords a good review from the French standpoint.

Directory of Pediatric Societies *

INTERNATIONAL

International Association of Preventive Pediatrics

President: Professor Taillens, Lausanne, Switzerland.
Secretary: Dr. P. Oltramare, rue Decandolle, Geneva, Switzerland.

FOREIGN

Royal Society of Medicine, Section for the Study of Disease in Children

President: Dr. Leonard G. Parsons, 58, Calthorpe Road, Birmingham, England.
Secretary: Dr. W. G. Wyllie, 33, Wimpole St., W. 1, London.

NATIONAL

American Medical Association, Scientific Assembly, Section on Diseases of Children

Chairman: Dr. Alfred A. Walker, Highland Plaza Apartments, Birmingham, Ala.
Secretary: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.
Place: Cleveland. Time: June 11-15, 1934.

American Academy of Pediatrics

President: Dr. John Ruhräh, 11 E. Chase St., Baltimore.
Secretary: Dr. C. G. Grulee, 636 Church St., Evanston, Ill.
Place: Cleveland. Time: June 11-12, 1934.

American Child Health Association

President: Dr. Samuel McC. Hamill, 1822 Spruce St., Philadelphia.
Secretary: Dr. Philip Van Ingen, 125 E. 71st., New York.

American Hospital Association, Children's Hospital Section

President: Mr. Robert Witham, Children's Hospital, Denver.
Secretary: Miss Agnes O'Roke, Kosair Crippled Children's Hospital, Louisville, Ky.
Place: Philadelphia. Time: Sept. 24-28, 1934.

American Pediatric Society

President: Dr. Charles A. Fife, 2104 Spruce St., Philadelphia.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid, St. Louis.
Place: Asheville, N. C. Time: May, 1934.

Canadian Society for the Study of Diseases of Children

President: Dr. H. P. Wright, 1509 Sherbrooke St. W., Montreal.
Secretary-Treasurer: Dr. R. R. Struthers, 1414 Drummond St., Montreal.
Place: Kingston, Ontario. Time: June, 1934.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Society of Pediatric Research

President: Dr. Rustin McIntosh, College of Physicians and Surgeons, Columbia University, New York.
Secretary-Treasurer: Dr. Charles F. McKhann, Harvard Medical School, Boston.
Place: Atlantic City, N. J. Time: May, 1934.

SECTIONAL

North Pacific Pediatric Society

President: Dr. Arthur H. Gray, 1117 Second Ave., Seattle.
Secretary: Dr. Raymond H. Somers, Cobb Bldg., Seattle.

Southern Medical Association, Section on Pediatrics

Chairman: Dr. Hugh L. Dwyer, 406, W. 34th St., Kansas City, Mo.
Secretary: Dr. Luther W. Holloway, 117 W. Duval St., Jacksonville, Fla.

STATE

Alabama Pediatric Society

President: Dr. C. E. R. Parker, 401 S. Court St., Montgomery.
Secretary-Treasurer: Dr. John W. Simpson, 1117 S. 22d St., Birmingham.

Georgia Pediatric Society

President: Dr. Joseph Yampolsky, 311 Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N. E., Atlanta.
Place: Augusta. Time: May, 1934.

Louisiana State Pediatric Society

President: Dr. John Signorelli, Louisiana State University Medical School, New Orleans.
Secretary-Treasurer: Dr. Edwin A. Socola, Louisiana State University Medical School, New Orleans.
Place: New Orleans. Time: April, 1935.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. Adolph G. De Sanctis, 12 E. 11th St., New York.
Secretary: Dr. George M. Retan, 459 James St., Syracuse.

Michigan State Medical Society, Pediatric Section

Chairman: Dr. W. A. Collins, 103 W. Burdick St., Kalamazoo.
Secretary: Dr. Edgar E. Martmer, 749 David Whitney Bldg., Detroit.
Place: Battle Creek. Time: September, 1934.

Mississippi State Pediatric Society

President: Dr. N. C. Womack, Jackson Infirmary, Jackson.
Secretary-Treasurer: Dr. Guy C. Jarratt, 920 Crawford St., Vicksburg.
Place: To be designated by Society. Time: Yearly.

Nebraska Pediatric Society

President: Dr. W. O. Colburn, 725 Sharp Bldg., Lincoln.
Secretary-Treasurer: Dr. C. G. Weigand, 1614 Medical Arts Bldg., Omaha.
Place: As announced by committee. Time: Second Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

North Carolina Pediatric Society

President: Dr. J. Buren Sidbury, 15 N. Fifth St., Wilmington.
Secretary: Dr. E. K. McLean, 121 W. Seventh St., Charlotte.
Place: Various children's hospitals. Time: Twice a year.

OKLAHOMA STATE PEDIATRIC SOCIETY

President: Dr. Clark H. Hall, 117 N. Broadway, Oklahoma City.
Secretary: Dr. M. J. Searle, 108 W. 16th St., Tulsa.

SOUTH CAROLINA PEDIATRIC SOCIETY

President: Dr. Julian P. Price, Medical Bldg., Florence.
Secretary-Treasurer: Dr. Leland B. Salters, 317 S. Warley Ave., Florence.

TENNESSEE STATE PEDIATRIC SOCIETY

President: Dr. Owen H. Wilson, 706 Church St., Nashville.
Secretary: Dr. Joe T. Smith, 702 Medical Arts Bldg., Knoxville.

TEXAS PEDIATRIC SOCIETY

President: Dr. Sidney R. Kaliski, Moore Bldg., San Antonio.
Secretary-Treasurer: Dr. Frank M. Martin, 321 Medical Arts Bldg., San Antonio.

VIRGINIA PEDIATRIC SOCIETY

President: Dr. Thomas D. Jones, Medical Arts Bldg., Second and Franklin Streets, Richmond.
Secretary: Dr. W. A. McGee, 2618 Grove Ave., Richmond.
Place: Alexandria. Time: October, 1934.

WEST VIRGINIA STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

President: Dr. R. C. Hood, 428 W. Pike St., Clarksburg.
Secretary: Dr. Russell C. Bond, 1325 Chapline St., Wheeling.

LOCAL

ACADEMY OF MEDICINE OF CLEVELAND, PEDIATRIC SECTION

Chairman: Dr. J. A. Garvin, Carnegie Medical Bldg., Cleveland.
Secretary: Dr. William M. Champion, 2323 Prospect Ave., Cleveland.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

ACADEMY OF MEDICINE, TORONTO, SECTION OF PEDIATRICS

President: Dr. Gladys Boyd, 56 DeLisle St., Toronto.
Secretary: Dr. Lawrence Murray, 1870 Bloor St., W., Toronto.

BRONX PEDIATRIC SOCIETY

President: Dr. Morris Gleich, 9 Post Ave., New York.
Secretary: Dr. Harry D. Pasachoff, 1560 Grand Concourse, New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Every second Wednesday of each month, except June, July, August and September.

BROOKLYN ACADEMY OF PEDIATRICS

President: Dr. Carl H. Laws, 12 Pierrepont St., Brooklyn.
Secretary: John A. Monfort, 861 Carroll St., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Fourth Thursday of October, January and April.

Chicago Pediatric Society

President: Dr. Charles Schott, 48 W. Division St., Chicago.
Secretary: Dr. Bert I. Beverly, 8 S. Michigan Ave., Chicago.
Place: City Club. Time: Third Tuesday of each month.

Dallas County Pediatric Society

President: Dr. P. E. Luecke, 4105 Live Oak St., Dallas, Texas.
Secretary: Dr. R. S. Usry, 1835 Garrett Ave., Dallas, Texas.
Place: Bradford Memorial Hospital. Time: 1 p. m., first and third Saturdays of each month.

Detroit Pediatric Society

President: Dr. John A. Johnston, 2799 W. Grand Blvd., Detroit.
Secretary: Dr. Edgar E. Martmer, 749 David Whitney Bldg., Detroit.
Place: Wayne County Medical Society. Time: 8:30 p. m., first Wednesday of each month.

Fulton County Medical Society, Pediatric Section (Atlanta, Ga.)

Chairman: Dr. L. H. Muse, 384 Peachtree St., Atlanta.
Secretary: Dr. T. I. Willingham, 384 Peachtree St., Atlanta.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: Second Thursday of each month from October to April, 8 p. m.

Houston Pediatric Society

President: Dr. Frank Lancaster, Medical Arts Bldg., Houston, Texas.
Secretary-Treasurer: Dr. A. A. Little, Jr., 817 Holman Ave., Houston, Texas.
Time: Third Monday of each month.

Intermountain Pediatric Society

President: Dr. Helmina Jeidell, 11 Exchange Pl., Salt Lake City, Utah.
Secretary-Treasurer: Dr. Edwin R. Murphy, 9 Exchange Pl., Salt Lake City, Utah.
Place: Salt Lake City, General Hospital. Time: First Thursday of each month, 8 p. m.

Kansas City (Missouri) Pediatric Society

President: Dr. Hugh L. Dwyer, 406 W. 34th St., Kansas City.
Secretary: Dr. John Aull, 906 Grand Ave., Kansas City.

Memphis Pediatric Society

President: Dr. Harry J. Jacobson, 1193 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. Clyde V. Croswell, 1052 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

Milwaukee Pediatric Society

President: Dr. F. R. Janney, 1411 Wauwatosa Ave., Wauwatosa, Wis.
Secretary: Dr. John H. Reynolds, 1628 Wisconsin Ave., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

New England Pediatric Society

President: Dr. James L. Gamble, 33 Edgehill Road, Brookline, Mass.
Secretary: Dr. Gerald Hoeffel, 319 Longwood Ave., Boston.
Place: Boston Medical Library. Time: Four meetings a year, occurring from September to May.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Martha Wollstein, Babies' Hospital of City of New York, New York.
Secretary: Dr. Harry Bakwin, 132 E. 71st St., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northwestern Pediatric Society

President: Dr. Alexander Stewart, 459 Lowry Medical Arts Bldg., St. Paul.
Secretary: Dr. E. F. Robb, 1801 Lyndale Ave., S. Minneapolis.

Philadelphia Pediatric Society

President: Dr. John D. Donnelly, 115 Bryn Mawr Ave., Bala, Pa.
Secretary: Dr. James E. Bowman, 4091 Comly St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

Pittsburgh Pediatric Society

President: Dr. Henry C. Flood, 119 S. Highland Ave., Pittsburgh.
Secretary: Dr. E. R. McCluskey, Highland Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate months from October to April, inclusive.

Richmond Pediatric Society

President: Dr. W. A. McGee, 2618 Grove Ave., Richmond, Va.
Secretary-Treasurer: Dr. Emily Gardner, 1100 W. Franklin St., Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: First Thursday of each month (except July, August and September).

Rochester Pediatric Society

President: Dr. H. F. Rowley, 176 S. Goodman St., Rochester, N. Y.
Secretary: Dr. J. M. Parker, 26 S. Goodman St., Rochester, N. Y.
Place: Arranged by program committee. Time: Second Friday of each month from October to May.

Rocky Mountain Pediatric Society

President: Dr. Arthur L. Esserman, Republic Bldg., Denver.
Secretary: Dr. Ralph Verploeg, 19th and Gilpin Sts., Denver.
Place: Children's Hospital, Denver. Time: First Saturday of each month from September to May, inclusive, 7 p. m.

St. Louis Pediatric Society

President: Dr. Hugh McCulloch, 325 N. Euclid Ave., St. Louis.
Secretary-Treasurer: Dr. Katherine Bain, Beaumont Bldg., St. Louis.
Place: St. Louis Medical Society. Time: Third Friday of every month.

Seattle Pediatric Society

President: Dr. Armin C. Rembe, 1215 4th Ave., Seattle.
Secretary: Dr. Frank H. Douglass, Medical-Dental Bldg., Seattle.
Place, College Club. Time. Third Friday of each month, 7 p. m.

Southwestern Pediatric Society

President: Dr. James E. Harvey, 60 S. Lake Ave., Pasadena, Calif.
Secretary: Dr. E. Earl Moody, 3780 Wilshire Blvd., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. John F. Sanders, 320 Townsend St., Lansing, Mich.
Secretary: Dr. D. Murray Cowie, University of Michigan, Ann Arbor, Mich.
Place: University Hospital, Ann Arbor, Mich. Time: Nov. 23-24, 1934.

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